THE

AMERICAN JOURNAL
OF THE MEDICAL SCIENCES.

NOVEMBER, 1906.

SPECIAL ARTICLE.

THE DIAGNOSIS OF RENAL AND URETERAL CALCULI.

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Stone in the kidney and ureter is a dangerous and painful malady. Tenney[1] says in 3807 autopsies in the Boston City Hospital, stone in either the kidney or ureter was found 21 times, and gallstones 146 times. During the same period he found there had been admitted to the hospital wards 109 cases of kidney or ureteral calculus and 489 cases of gallstones. These data show that the proportion (1 to 7) of urinary calculi that were not troublesome was very small as compared with gallstones, while in the patients admitted for these conditions the proportion was one to four and a half.

There are perhaps few questions of greater perplexity than the differential diagnosis of several pathological or abnormal conditions of the kidney and ureter and adjacent structures. Of these conditions probably none furnishes more difficulty than renal and ureteral calculus, though a few radiographers state that all doubt is swept away by the Röntgen rays. Whether this enthusiastic declaration is beyond question has not been determined by the large army of surgeons who have especially studied the subject of calculi of the urinary tract. Surely the time has not been reached when a careful analysis of the protean symptoms and previous history of these conditions seems superfluous. The careful study of cases of calculi in these structures will always be deemed of great importance if we can rely upon the painstaking and systematic urinary surgeon. That

[1] Boston Med. and Surg. Jour., 1902, cli, 660.

such study is conclusive and satisfactory in all cases is controverted by the many recorded failures to find calculi by operative exploration.

Symptoms. We may recognize three groups of symptoms, two of which may be called representative of acute attacks, and the third of a chronic condition. In the first one, the patient may be suddenly seized with intense pain in the region of the kidney or ureter that doubles him over or causes him to fall. If in bed at the beginning he feels unable to arise; vomiting soon supervenes, frequent micturition with a deficient amount of urine is noted, and delirium may rapidly occur. The pain, if the stone be in the ureter, may radiate into the bladder, urethra, testicle, or labium. Sometimes it is reflected even to the heel. Retraction of the testicle is more readily noticed in childhood owing to the tonicity of the cremaster muscle and the little weight of the testicle. In spite of prodigious doses of morphine the suffering is intense and remains so for from two to fifty hours. Involuntary discharge of feces and urine often occur. A few hours after the inception of the attack blood is discovered in the urine; it may continue for a few days. For a short time, too, following subsidence of the pain, a localized soreness is manifest.

In a second class of acute or recurrent cases the patient has frequent attacks of renal crises that are not so severe and seem to be expected by him. They usually occur after some vigorous exercise, such as playing tennis or running. At first there is a feeling of discomfort and then of localized pain in the affected side; vomiting, fainting, and loss of sphincteral control follow, and great prostration occurs. The pain may extend, in ureteral cases, to the corresponding side of the bladder. The quantity of urine is decreased during the attack, but increases subsequently. It may be normal in composition. A tumor in the region of the kidney may appear, though this is less common when the stone is in the kidney. The attack may last but a short time and recur at short intervals.

In the third or chronic class there is usually a history of neglected urinary calculus, though now there is only vague pain always localized in the urinary tract. It may radiate to the thigh, leg, or abdomen. The patient is not confined to bed, but has constant discomfort; variations in the quantity of urine are less marked, and even retention may occur. This class is most likely to be associated with infection, and the symptoms are correspondingly modified. The patient has a peculiarity in gait that indicates a desire to keep the psoas muscle relaxed. He sleeps on the affected side, and the pain may be greatest on the opposite side. Blood in the urine may be present, but not usually.

It is unfortunate that no positive symptom, other than the passage of the calculus, is known. Could reliance be placed upon the combination of any two or even three symptoms great assistance would be afforded the surgeon in very many instances. Indeed, many times

a stone in the kidney or ureter has reached a large size without attracting in any way the attention of the patient. This has been ascertained by autopsy and by an attack of renal colic being promptly followed by nephrolithotomy or ureterolithotomy, by which the character of the stone was ascertained.

The principal symptoms of renal or ureteral calculus are pain, hematuria, urinary changes (both in quantity and quality), and frequent urination. Beside these, various others are ofttimes present and quite as often increase as lessen the difficulty of differentiation. Of these may be mentioned the presence of a mass in the region usually occupied by the kidney—which may be a kidney much disturbed by calculi, hydronephrosis, or pyonephrosis. Fever, a chill, a history of previous attacks, the presence of a stone in the bladder, or the history of the passage of one or more of stones, or of sand, are commonly met with. These are of great assistance in diagnosis.

Pain is a symptom very variable. It depends largely upon the location of the stone in the kidney and its mobility. If in the parenchymatous or cortical portions pain, if present, is not prominent. If in the pelvis the pain becomes intense, in fact, produces what is said to be the most severe pain known. If it completely obstructs the outlet of the pelvis of the kidney acute hydronephrosis occurs, and this increases the pain. It is more than probable that the pain in renal colic is distinctly local, though the reno-renal reflex is often observed in the fellow kidney. It is of the sharp, lancinating variety and is said to radiate downward along the urinary tract, or down the thigh by way of the gluteal muscles. Sometimes the radiation is toward the umbilicus. I am confident, however, and my experience is largely the basis of this opinion, that such radiations are due to ureteral involvement. If the obstruction is not complete chronic hydronephrosis results and the pain is more constant and less severe. It may be entirely absent except by causing trouble in neighboring structures from the encroachment incident to the large size of the mass. Even when anuria has resulted from permanent obstruction by a calculus, pain may entirely subside and the calculus remain quiescent for years. Instances of marked renal atrophy resulting from calculous anuria are not wanting in the literature of this subject. In frequent slight attacks of renal colic the pain arises markedly. The size of the calculus seems to have no relation to the degree of pain; the composition of the calculus, however, does seem to have such influence, the oxalate or mulberry stones appearing to cause the worst attacks, and the uric acid stones the least severe.

Hematuria is an important symptom, since rarely does a calculus become restless without causing hemorrhage. It may vary from a few blood cells discoverable microscopically in the urine to a quantity sufficient to alarm the patient and the surgeon. Death from calculous hematuria has occurred. When associated with other symptoms of renal calculus bloody urine is of marked signi-

ficance; alone it is of much less importance. Slight increase in blood pressure and enfeebled capillary walls lead to slight kidney hemorrhage. It is very common in papilloma and other neoplasms of various portions of the urinary tract. In tuberculosis it is a fairly common symptom. It is a usual symptom of traumatism of the kidney or bladder. In calculus it may be absent or it may be the only symptom; Cunningham[1] found it in 22 of his 48 cases. It may be said, however, that when due to calculus it is increased by exercise, is most likely to accompany or quickly follow colic, and rapidly subsides when the patient is made to rest in the horizontal position. The clots are long and perhaps of irregular shape, being molded about a calculus or in the ramifications of the kidney pelvis. They may be four or five inches in length. The terminal end is usually very long and slender. They are best studied after washing through many waters so that they are transparent.

The urinary changes in calculus are not constant, but very valuable information is secured from systematic routine urine analyses. In calculus of the urinary tract the urine may be entirely normal; this is most likely to be the case with quiescent uric acid calculi, yet the urine is almost certain to contain minute fragments of the stone, pus, or an unusual amount of epithelium from the involved part. Cunningham found pus in 39 of 48 cases. After attacks of colic from stone we may expect to find blood in the urine, varying from mere traces to large clots. If the stone completely blocks a ureter urinalysis will furnish no information of the conditions in the urinary tract above the obstruction.

Frequency of micturition occurs when the stone is restless in the ureter, and the intensity of the pain corresponds to the nearness of the stone to the bladder. (A woman patient of mine had a quiescent calculus in the lower part of the ureter, more than one inch in its smallest diameter, that produced no bladder irritability nor stoppage of urine from that side.) As a rule, uncomplicated renal calculi do not produce marked vesical irritability.

The presence of a mass in the usual location of the kidney may be expected after an attack of unilateral ureteral colic, though it is by no means always present. If acute ureteral obstruction has been produced by the calculus, dilatation of the urinary tract proximal to the site of obstruction will occur, and, within certain limits, will correspond to the duration of the obstruction. It never reaches the size that results from intermittent obstruction from kinks. If pyonephrosis is marked the enlargement is greater than in acute hydronephrosis and the mass is less elastic.

A *chill* may result from the impress upon the nervous system from pain or from toxemia. *Fever* indicates pyonephrosis, and a leukocyte count at this time will show a varying leukocytosis. Tenney saw it rise in four hours from 12,000 to 16,000.

[1] Boston Med. and Surg. Jour., 1902, clii, 655.

The history of previous attacks of calculous colic is not always attainable. Perchance there has been none. However, as a calculus in passing through the urinary tract is very likely to find various lodgements causing colic, a strong probability exists that attacks have occurred or will occur. If to these symptoms we may be able to add the fact that a stone has been passed *per urethram* or exists in the bladder, we have a group of symptoms that justify a diagnosis of renal or ureteral calculus. That such calculi may be bilateral should be borne in mind.

In the combined tables of Cunningham and Morris[1] the right side was involved 119 times, the left 95, and both 11 times. Necessarily the 5 per cent. of bilateral cases may cause much confusion in diagnosis, unless bilateral anuria should be produced. In cases of unilateral calculous anuria, complete anuria from blocking of the fellow ureter gives the surgeon great concern as to which side should be selected for operation. Palpation may find the silent kidney atrophic and the functionating one hydronephrotic. In that event the diagnosis is established. The limits of age in which renal and ureteral calculi are found is practically twenty and forty years. Beyond forty they are not commonly found, but this cannot be said of the first twenty years of life. Cabot[2] found the age varied in his cases from fifteen to fifty-four years, the average being thirty years. Sex seems to have no significance, as the combined tables of Tenny, Morris, and Tuffier show it to have occurred 276 times in women and 259 times in men. Probably the surgeon who treats more women than men finds it oftener in women, and *vice versa*.

Diagnosis. The various features of physical examination that are of moment are drinking large quantities of water, palpation, including rectal, intravesical, and vaginal palpation, cystoscopy, ureteral exploration with catheter, probe, or waxed tipped bougie, skiagraphy, and exploratory incision. Brewer[3] and others found that making the patient drink two or three quarts of water in a few hours forced calculi into the bladder. Palpation of the kidney rarely offers much information, though crepitation of calculi has been discovered. This must be a very rare occurrence. In thin subjects palpation through the abdominal wall of the upper two-thirds of the ureter might offer some information as to the presence of a mass, but probably under the most favorable circumstances the small calculi usually found in the ureter would thus escape such detection. Rectal palpation in the male and vaginal touch in the female offer very reliable information concerning calculus in the lower third of the ureter. Dilatation of the urethra sufficient for insertion of a finger for vesical or vesicoabdominal palpation will afford information as to the extreme lower end of the ureter. The danger of

[1] Surg. Dis. of Kid. and Ureter, 1901.
[2] Boston Med. and Surg. Jour., 1902, clii, 658.
[3] Annals Surg., 1904, li, 134.

permanent injury to the sphincter is so great that resort to it for diagnostic purposes alone should be extremely rare. The so-called Meltzer sign of increased pain by hyperextension of the thigh is noticeable in conditions other than this. The stamping test of Lucas[1] is of value in mild and doubtful cases, but I have found it severe in others.

The cystoscope is useful, as by it may be learned the condition of the vesical mucosa and the ureteral mouth, as well as whether a calculus is in the bladder. The principal value of it, however, is to ascertain whether both ureters are conveying urine and whether additional or abnormal ureteral mouths exist. Ureteral exploration is of great value, as a calculus in the ureter will nearly always be detected by it. Kelly's wax-tipped bougie will probably always show scratch marks when brought in contact with a stone in the kidney or ureter. The nephrophonotoscope of Eaton[2] and the phonophore of Follen Cabot,[3] nearly similar instruments, may prove unerring when brought in contact with such stone; but Lilienthal[4] mentions a failure of the latter instrument in the hands of Cabot and the subsequent successful ureterolithotomy above the pelvic brim. Obstructions from causes other than calculus are not differentiable by the ureteral catheter, but by its use, as with the segregator, the relative and absolute functional state of both kidneys may be learned. Bierhof[5] employed it for injecting boric acid solution into the kidney, claiming that if hematuria occurred within twenty-four hours a stone was present.

Exploratory incision is not justifiable except in calculous anuria, or when operation for some other condition is done, or when skiagraphy has failed to discover a calculus in the face of a very strong presumptive symptomatology. If the abdomen be opened for another purpose, intraperitoneal palpation, a very satisfactory procedure, may practically decide the diagnosis as to the ureter. The broad ligament portion of it, however, is not accessible to such palpation. Exploration of the kidney is best made by the longitudinal incision reaching through from the convex border to the pelvis. The entire ureter as well as the kidney may be explored from it.

At the present time skiagraphy is recognized by many as the *sine qua non* in the diagnosis of urinary calculi and its popularity is constantly increasing. The cases, now numbering hundreds, in which clearly depicted calculi have appeared in skiagrams and have later been removed by the surgeon have a strong tendency to convince the most skeptical. No doubt many poorly qualified skiagraphers are looking unintelligibly for renal calculi. They will not furnish

[1] Lancet, 1903, i, 1143–1148.
[2] Calif. State Med. Jour., 1905, iii, 357.
[3] Amer. Jour. Urol., 1905, i, 213.
[4] Internat. Jour. Surg., 1906, xix, 69.
[5] Med. News, October 11, 1902.

clear pictures nor will they be able to interpret them properly. They are not even well-informed concerning the literature of the subject. Were one to be guided by such results skepticism would continue, perhaps increase. Probably the most successful skiagrapher is C. L. Leonard, whose corrected statistics to February 1, 1905, stated by Tenney, are 93 cases with a positive shadow, and at operation stones were found in 87 of them, and 233 cases with negative shadows in which stones were passed subsequently or removed by operation in four instances. Hulst[1] says every kidney stone can be demonstrated by the Röntgen rays provided the technique is perfect. Rumpel claims 100 per cent. of correct skiagrams for renal calculi. Moseley[2] says radiography furnishes the only positive means of detecting renal or vesical calculi.

The most easily detected are those composed wholly of mineral salts, oxalate of lime, phosphates, and urates ranking in the order mentioned. Those made up wholly of organic matter photograph only with difficulty. Leonard[3] claims ability to get sufficient shadow from pure uric acid calculi. Fortunately these are rare. Boggs says even minute particles of uric acid may be detected. As has been mentioned much depends on the technique of the operator and upon the proper reading of the radiogram. The plate should be large enough to photograph both kidneys, both ureters, the lower ribs, and the psoas, iliacus, and quadratus lumborum muscles. Satisfactory negatives should show the outlines of the vertebral bodies, the intervertebral spaces, the transverse and spinous processes, as well as a fine differentiation between structures. Several pictures should be made—from both the front and side. Great care is necessary also in selecting the plates that defects in them may be avoided.

No doubt errors have arisen from shadows of substances near the ureter. The cases cited by Edebohls[4] and myself were of this character. Lilienthal mentions sesamoid bones in the internal obturator muscle, calcified lymph nodes, thick lime plates in the internal iliac artery, and foreign mineral bodies in the rectum, and Brewer mentions phleboliths as producing such conflicting shadows.

When the history of the patient has been carefully secured and analyzed and the methods of non-operative examination conducted thoroughly, if doubt remains as to the presence of calculus in the kidney or ureter, resort to radiography should be made. When carefully done it should afford the data to complete the record from which a diagnosis is to be made. It is the most reliable of all methods of examination, yet the extreme position of Leonard and a few other

[1] Amer. Med., 1904, viii, 580–1100.
[2] New York Med. Jour., 1903, March 7, 415.
[3] Annals Surg., 1901, xxxiii, 435–442.
[4] Trans. Amer. Gyn. Soc., 1904, xxix, 89 and 91.

very expert radiographers is not maintained by a large number of the most experienced and progressive surgeons of the age.

DIFFERENTIAL DIAGNOSIS. Of the many conditions that make the diagnosis of calculus of the ureter and kidney so uncertain mention should be made of tuberculosis of those structures, hydronephrosis (especially from kinks of the ureter), pyonephrosis, renal neoplasms, acute nephritis, broad ligament tumors, ectopic pregnancy, fused kidney, double ureter, hepatic disease, appendicitis, intestinal new-growth, gallstones, concretions in the pancreatic duct, and caries of the spine.

Tuberculosis in the acute stage may furnish few differential symptoms. Here again the necessity of careful and multiple urine analyses is demonstrated. Tubercle bacilli may be discovered in the urine and the tuberculin and inoculation tests are available. In later stages the same tests are applicable, and in addition the large cord-like upper ureter is demonstrable, especially under anesthesia. Later, the lower portion may also be found to be similar. Debris containing the tubercle bacilli may be found in the urine. Perhaps the disease may be detected in other tissues.

Hydronephrosis from other causes will not produce abnormality of the urine, except a deficiency in solids. In the chronic form it does not afford the symptoms of pain and hematuria. When due to angulations or kinks of the ureter it usually gives rise to colicky pain and the train of symptoms significant of renal crisis. The history of deficient quantity of urine, associated with constantly increasing pain and the development of a renal tumor, and the rapid reversal of these symptoms, particularly when relief comes from change of position, offer great differential data. Pyonephrosis does not furnish such clear points of distinction. Indeed, most cases of this condition originate in renal calculus.

Renal neoplasms give rise to pain that is steady, and the development is gradual and progressive. An exception to this is hypernephroma which after considerable development may remain quiescent for long periods of time, again to take on rapid increase in size. Most renal neoplasms are malignant and cause death in a few months. Papilloma is especially likely to produce troublesome hematuria.

Acute nephritis may cause severe pain on both sides and anuria, but it does not cause colic, and the urine affords differential details. Broad ligament tumors and enlarged or calcareous pelvic lymphatic glands may be mistaken, on palpation, for stone in a related portion of the ureter. Pressure of them against the ureter often causes vesical irritation. Here again the history of the case with urine analysis will usually lead to differentiation. In some cases ureteral exploration or even radiography may be required.

Ectopic pregnancy in the first or second month may be confounded with ureteral colic. Usually, however, urine analysis, a searching

history inquiry, and a careful physical examination, perhaps including the interior of the uterus, will permit a clear distinction being made. Fused kidney and kinked ureters offer unusual difficulty in differentiation from renal or ureteral calculus. In this condition the kidneys are in front of the lumbosacral junction and the ureters usually join the kidney at a sharp angle. Calculi in this condition cause localized pain that is confounded with disease of the intestine or of the female pelvic organs; ureteral exploration is confusing, as the ureters are about half their usual length and the renal pelvis is not so amenable to exploration. Many times the diagnosis will be made only by the radiogram or exploratory incision. In double ureter the calculus may be in one of them occluding it, while the other is permeable throughout its length. When this occurs in the ureter that is forked above, ureteral exploration will render but little aid. Reliance for diagnosis must be placed upon the symptoms, urine analysis, and the skiagram. Hepatic disease should be confounded with renal calculus only in so far as a tumor of the liver or distended gall-bladder may be mistaken for an extraperitoneal mass in or about the kidney, and hepatic colic mistaken for renal colic. Usually, such symptoms as jaundice and marked digestive disturbances permit a distinction. If not, urine analysis and ureteral exploration, or, possibly, radiography, will clarify the situation. Many times ureteral calculi have caused a diagnosis of appendicitis to be made. This is scarcely pardonable, however, as the symptoms are commonly at wide variance.

Lucas furnishes an interesting instance of spinal caries being mistaken for renal calculus. He was called to operate and found the child suffering with unilateral pain in the back radiating downward. Incision and drainage proved sufficient. A few days later the same diagnosis was made for the other side. By telegraph he urged searching for a similar abscess on the painful side. It was found and evacuated with relief.

ORIGINAL ARTICLES.

THE ENVIRONMENT IN THERAPEUTICS FROM THE STANDPOINT OF PHYSIOLOGY.[1]

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The object of the present paper is to review briefly a few of the elements of environment in their relation to the treatment of disease, and to point out some of the ways in which modern therapeutic

[1] Read at the meeting of the Association of American Physicians, Washington, D.C., May 15 and 16, 1906.

practice, especially in hospitals, through neglect of these considerations, sometimes fails to realize all that it might.

With the development of cellular pathology and the discovery of anatomical and bacteriological causes of so many diseases has come an inevitable and useful spirit of materialism in medicine, and a tendency to consider and to concern one's self only with tangible matters—those that can be seen and touched. This has been a useful spirit, and necessary to the transition from the old fantastic medicine of the past to the medicine of the present imbued with and founded upon the principles of science. But with it something has been lost that was of use.

Consideration for the surroundings of the patient has come to be regarded as belonging rather to the care than to the treatment of disease, and so has been left to the nurse. The development of the trained nurse system, too, has contributed to this result, and physicians have often grown unfamiliar with the management of the details of the surroundings of their patients.

But gradually confidence in the potency of many drugs has weakened. Certain of these agents, to be sure, have maintained their position, and probably always will maintain it; but the number of diseases in which we sum up the treatment as consisting of good care and the treatment of symptoms has steadily grown. This list of diseases promises for a long time to be an extensive one, to be shortened only as modern scientific medicine gradually develops specific forms of treatment.

It seems fair, then, every now and then to review the subject of environment in therapeutics, and to examine it in the light of the most recent teachings of physiology, pathology, and experimental medicine, and to see whether it cannot be made worthy of the earnest consideration of the thoughtful physician. It is a very old subject, and one to which much thought has always been given.

Sydenham regarded horseback exercise in the open air as a specific for consumption, and many years ago the open-air treatment of pneumonia was advocated, while mental influence has always been an important part of the armamentarium of every successful physician. The psychical side of environment has long been made use of by neurologists, but mainly in relation to functional neuroses. In a recent work, and one of the most useful that has lately appeared, on "The Psychical Treatment of Nervous Disorders," Dubois makes a powerful and unanswerable plea for the formal consideration of the psychical environment of the individual in many or in most disorders. This work, being based not upon empiricism wholly, but in very large part upon the teachings of modern physiological psychology, deserves the earnest consideration, not only of the neurologist, but of every person who is called upon to care for the sick and injured. It is by no means a new principle in therapeutics, but an old one that needed to be formulated, and whose

place in treatment required to be defined and accentuated; this Dubois has done in a masterly manner.

The use of the psychical side of environment in treatment is limited to no single group of diseases. In the neuroses or in other disorders in which neuroses play a considerable part it must be one of the main dependences; and this principle, as is so well elaborated by Dubois, no longer rests upon empiricism, or upon vague, undemonstrable theories of mental influence, but is firmly founded upon established principles of psychical physiology, both normal and morbid.

But in every case of disease the psychical state of the individual is an important element in maintaining and aiding the reaction of the tissues to any injury, whether of parasitic, metabolic, or other origin. Just as the influence of the environment upon the nervous system is an important element in causing disease, so it can be made equally important in combating disease, not only the neuroses but all organic maladies.

Celsus says "A great many of the inhabitants of cities and towns, and almost all the lovers of learning, have weak stomachs." And then follows a fantastic explanation of this true observation, based upon the faulty idea of physiology, pathology, and psychology current at his writing. Many centuries after, Ramazzini repeated the observation, and also used the same explanation; but now the modern teachings of the anatomy and physiology of the nervous system, and of psychology, and the relation of these elements to one another, have given us a satisfactory scientific explanation of the frequency of digestive neuroses in the classes of persons referred to. Like Celsus, we recognize the frequency of the condition and its dependence upon environment, and we find that modification of this element is the most important single means that we possess in combating the majority of digestive disorders; but the modification of environment from a psychical standpoint belongs to the treatment of every disease, and when it cannot cure, it can often aid other means to either cure or ameliorate.

In hospitals, unfortunately, it is more difficult of application, and as a rule, it is completely neglected. I have seen a clinic in one of the universities of Europe in which the viscera of a patient that died the preceding day were demonstrated and explained in the presence of another patient with the same disease waiting his turn to be lectured upon.

Few hospitals are so arranged that those who are dying shall not be seen or heard by patients in neighboring beds, or so that the psychical side of the environment of the individual shall have any consideration whatever. A few surgical hospitals have recovery rooms in which operative cases can be cared for in some isolation while recuperating from anesthesia, but even here it has generally been done with consideration only for the patients operated upon, and not for the remaining patients in the ward.

In most institutions recovery from anesthesia takes place in the wards, and surely the sight and sounds of the procedure are but poor preparation for similar operations on others. Many other instances might be given of the failure to appreciate the importance of the psychical side of environment in the designing, equipping, and management of hospitals that are supposed to be modern. It is much to be desired that architects, trustees, and others concerned in planning public institutions should give due weight to this important feature, and should realize that it rests not as formerly upon the vague and uncertain ground of hypothesis, but upon the certain ground of the teachings of modern physiological psychology.

Probably the psychical value of segregation is not always clearly understood. The value of sanatorium treatment of many neuroses depends partly upon the association of the patient with others of the same class.

In pulmonary tuberculosis there is a distinct advantage in sanatorium treatment or in treatment in colonies, which cannot be explained entirely on the ground of discipline and methodical and regular life. Contrary to the general belief of the laity, these patients as a rule do best in tuberculosis colonies. The restrictions placed upon their activities and appetite are less irksome, and they fall with more readiness and cheerfulness into a hygienic routine than when they stand alone. They appear to be under somewhat the same psychical influence as soldiers who march farther and with less complaint and exhaustion when in company formation and keeping step than when straggling alone.

One finds the same indefinable but real influence of environment in persons undergoing treatment at health resorts, such as Carlsbad, where the early rising, the nauseous draughts of waters, and the rigid dietetic restrictions are submitted to with the utmost cheerfulness in company, when only open rebellion would often result were the individual asked to go through the same regimen alone.

These will serve as examples of some of the ways in which the psychical side of hospital treatment will bear investigation and modification; and this investigation is all the more indicated, since failure so to consider the environment, cannot be regarded as merely failure to use a measure for good but as perpetuation of conditions that are positively a hindrance to cure.

The physical side of environment includes: (1) Temperature; (2) ventilation; (3) barometric pressure; (4) humidity; (5) light; (6) quiet, noise, etc.

It is not my purpose to take up each of these in detail. Some, as barometric pressure and humidity, do not admit of general artificial variation. The application of others is too obvious to justify discussion. That cheerfulness in the sick-room is to be preferred to gloom, quiet to turbulence, fresh air to a close atmosphere, does not permit of argument; but there have recently been

developed certain general therapeutic tendencies along these lines that promise well enough to justify discussion and wider publication; and there are suggestions that can be made looking toward a more thorough consideration of the question of the physical side of environment, especially from the hospital standpoint, and in relation to physiology, that seem to contain possibilities for much good.

With the discovery of the cause of tuberculosis, has come a thorough rehabilitation of the therapeutics of the disease, together with a conviction of the utter worthlessness, even injuriousness, of most of the older forms of medicinal treatment. Fresh air, feeding, and rest for the disease itself, and medicines only for special indications, and these as sparingly as possible; wherever the disease is well understood, these are the main points in treatment. These measures are based upon the soundest physiological principles. The invigoration of the body furthers the resistance of the tissues to the infection, and so favors cure. At first, the out-door treatment was attempted only in the pulmonary type, and the fresh air was admitted to be of use only because the disease was in the lungs. Then it was found that the non-pulmonary forms of tuberculosis showed just as striking benefit from out-of-door life as did the pulmonary, and now numerous successful institutions in various parts of the world testify to the fact that this is the most satisfactory method of treating tuberculosis of the bones, joints, lymph nodes, and various other parts of the body, where the disease is not susceptible of complete cure by operation. This conclusion, too, is justified by consideration of the physiology of respiration.

A young woman suffered from advanced tuberculosis of the kidney of many months' duration. As a concession to the impaired renal function she was kept closely housed in a warm room maintained at an even temperature, and she was with the same purpose fed upon a restricted diet. She had continuous irregular fever, rapid pulse, progressive loss of weight and strength, and, indeed, all the evidences of an actively advancing tuberculous lesion. She then fell into other hands; the renal function was disregarded, and only the tuberculosis was considered; treatment adapted to pulmonary consumption, or, more properly, to tuberculosis in any part of the body, was instituted; it consisted entirely in rest out-of-doors in all weathers, and forced feeding. Almost at once the temperature became normal, active symptoms subsided, and the evidences of a quiescent tuberculous lesion were substituted for those of an advancing one.

No rational discussion of fresh air can be engaged in without the influence of temperature being considered at the same time. By the use of fresh air in the treatment of disease I mean something quite different from ventilation in the ordinary sense of the term. Most sick-rooms are very inadequately ventilated; many contain

air that is unfit to be breathed even by well persons, and I have never seen a modern, up-to-date hospital that seemed to me to be adequately supplied with fresh air. I believe that the so-called scientific tests of the quality of air in rooms are of only limited value, and cannot be used as a basis of final judgment; I mean, the estimation of the proportion of carbon dioxide and of the number of bacteria, or the amount and quality of dust present. Until some further methods of study are discovered the best test of the air in the sick-room is the sensation of an intelligent and alert physician on entering, or the sensations of an observant nurse after remaining there some hours; while the condition of the patient, his progress toward or from health, his restlessness, and his quiet or sleepless nights will help to a judgment.

In attempting to study the physiological bearing of ventilation, one is beset by the difficulty of determining what are the essential and effective elements of good and bad ventilation. All agree that there are certain well-defined symptoms that regularly appear when people remain in an atmosphere which is called close or stuffy, that is, one that has been occupied and breathed for a considerable time without the air being renewed. The principal symptoms are a feeling of heaviness and oppression, headache, drowsiness, malaise, vertigo, tinnitus aurium, nausea, and faintness. If the conditions are maintained for a longer time, there is loss of appetite, coated tongue, indigestion, nausea, constipation, and nervousness, and, subsequently, if the evil be persisted in, the general health may become seriously impaired, secondary anemia and malnutrition being pronounced features, together with a generally increased susceptibility to the infectious diseases.

It was long a matter of dispute whether these conditions were due to the fact that the air contained poisonous substances, given off from the body, or whether the increased amount of carbon dioxide was the essential feature.

Ransome[1] claimed to demonstrate toxins in the exhaled breath and in the water of condensation from such breath. Merkel[2] and Brown-Séquard and d'Arsonval[3] came to a similar conclusion. These poisons, which they called anthropotoxins, could not be isolated, but their existence was claimed to be proved by experiment. Most subsequent observers have come to the opposite conclusion, namely, that no such toxins exist in the air of expiration—Hermans[4] (confirmed by Rauer), Lubbert,[5] Kriegel, Mehl, Hermanek,[6] Billings, Weir Mitchell, and Bergey.[7]

More recently, under the direction of Flügge, Heyman, Paul, and

[1] Journ. Anat. and Phys., 1870, iv, 209.
[2] Arch. f. Hyg., 1892, xv, 1.
[3] Compte rendu, 1888, cvi., 106 & 165.
[4] Arch. f. Hyg., 1883, 1, 5.
[5] Ztschr. f. Hyg., l, xv.
[6] Arch. f. Hyg., 1900, xxxviii, 1.
[7] Smithsonian Contributions to Knowledge, 1895, vol. xxix, No. 989.

Ercklentz,[1] in an elaborate series of experiments upon men and animals, together with extensive chemical tests, confirm the results of the latter group, and declare that the anthropotoxins have no existence. They find that the deleterious effects of close atmospheres consist, not in the carbon dioxide, but in the elevated temperature, humidity, and absence of air currents. Paul placed men in small, close chambers until the atmosphere became very impure, but kept the air in active motion by fans, and found that no symptoms were developed. He then placed them in a similar environment but with the air still, and had them breathe pure fresh air through tubes. Here the regular symptoms of bad ventilation occurred, but were dissipated when the same air was put in active motion.

Flügge concludes that the sensation of well-being experienced with good ventilation is due to the abstraction of heat from the body through the evaporation of moisture from the surfaces, and that the removal of expired air plays a relatively unimportant part. Hence, he concludes that the features to be sought after in the atmosphere are coolness, dryness, and active motion. He considers the CO_2 content relatively unimportant. Rubner finds that it takes but slight motion of the air markedly to increase heat loss.

It has been found difficult to establish the point at which CO_2 in the atmosphere becomes a distinct injury to health. Paul found that animals died when CO_2 reached 14 per cent., and that it was immaterial whether this was produced by the respiration of other animals or by the action of chemicals. The atmosphere of large cities contains about 4 parts of CO_2 in 10,000; that of the open country about 2.5 parts. It has been generally assumed that in rooms it should never go above 10 parts. Flügge's conclusions point to the unreliability of CO_2 determinations in deciding whether an atmosphere is fit for human beings. He seems to conclude that many atmospheres may be good enough to be breathed which are not good enough to belived in.

His experiments and conclusions carry much conviction with them, and suggest some valuable points in the management of the environment of the sick, such as the use of fans, and the attempt to secure lower temperature and more motion in the air.

It is a matter of practical experience that most modern artificial ventilation systems are satisfactory only to the architects and engineers who have installed them; they are entirely unsatisfactory to those who must live in them.

In view of the above conclusions from experiment it seems probable that this results because the ventilation is devised purely from the standpoint of respiratory requirements, based upon the oxygen needs of the individual, rather than from the cutaneous needs.

[1] Ztschr. f. Hyg. u. Infek., 1905, xlix, 433.

I have had noted and charted the humidity of wards of different type, and at the same time that of the outside air. Even with windows wide open, the humidity in the ward is much above that of the exterior, while it rises still higher proportionately to the number of occupants, and inversely according to the number of windows that are open. These differences are greater in cold weather, but are also present when the temperature outside and within are about alike.

In the modern system, moreover, special effort is made to avoid sensible air currents, while in Flügge's experiments such currents were one of the chief factors in relieving the symptoms resulting from a close atmosphere. In practice in the sick-room and the ward I have always found draughts a far less serious bugbear than they are generally considered to be.

I have put these principles in operation in the hospital wards, and while sufficient time has not yet elapsed to enable me to give numerical results, yet the outcome up to the present has been encouraging and sufficient to justify the continuance of the experiment.

Moreover, I suggest the possibility that air that has been drawn through long, closed ducts, and then superheated over coils of steampipe has lost certain qualities that belong to fresh air. This suggestion is a vague one, I admit, and is not susceptible as yet of scientific demonstration, but is based upon actual experience in hospital wards, and the observation of the difference in the progress of patients in closed wards supplied with a modern artificial ventilating system, and in the same wards after the ventilating system has been cut off and the windows opened.

Canned foods contain the same chemical substances as do the same material when fresh, but they are less well suited to constant, continuous human consumption.

There has long existed a tradition that the sick-room or ward should be maintained at a constantly even temperature, or as near such as may be possible. I should like to know the origin of this tradition, and whether it is founded upon theory or experience. Surely in health the body finds an even temperature far from grateful, and, on the contrary, craves the alternations which, in most parts of the world, are supplied throughout the year by the changing seasons, and through the day by the changing relation to the sun. There are diseases, to be sure—for example, certain cases of nephritis, in which extreme changes of temperature are probably injurious, and in which exposure to extreme cold is to be avoided, although even here one may question whether we have not been overafraid of such influence.

Dr. Northrup has recently seen a case of acute nephritis in a child show marked and rapid improvement, and go on to recovery, when removed from the hospital ward and placed upon the open

roof of the building in midwinter. This, to be sure, is an experiment, and is not to be recommended at present for general adoption. In this case the environment was changed because the child was doing badly and showed unfavorable nervous symptoms.

I have, however, tried the effect of marked alternations in the temperature of sick-rooms and wards, opening the windows wide in midwinter, and have found only improvement to result. This improvement, it is true, has followed the combination of change in temperature and the admission of fresh air, and I have concluded from it, as far as temperature itself is concerned, merely that in most cases there is no advantage to be had from the maintenance of the sick-room at an even or at any fixed temperature.

The experiment has been tried in medical wards containing such patients as are generally to be found in any acute hospital, and thus far has indicated that except in cases of advanced disease of the kidneys and heart, and with consideration for the personal comfort of the patient, the temperature of the room or ward may be disregarded.

At present, as a rule, the temperature is adopted to meet the comfort of thinly clad nurses, or, in private practice, of some elderly relative.

In most hospitals the rule obtains that the wards are to be kept at as near as possible to 70° F. both day and night; thus, as far as treatment by this feature of environment is concerned, forcing all patients alike into one class, no matter what the nature of their disease may be.

The influence of good ventilation in pneumonia has long been recognized, and is insisted upon by all who write upon this disease, but the idea has never become popular with the laity, though recently it is growing easier to obtain a reasonable supply of air for the patient when one is dealing with intelligent people. The majority of the medical profession are equally ignorant, however, and sin in the same direction. But even good, normal ventilation is not sufficient for a patient with pneumonia. He should have fresh air; as near as possible an approach to out-door treatment, unless there is some special contraindication.

Von Leyden[1] devotes thirty-two pages to a masterly discussion of the treatment of pneumonia, but dismisses the question of environment with but a few scanty and insufficient words. Most textbooks preface the section on the treatment of pneumonia with the statement that the room should be kept at an even temperature. I have demonstrated by abundant experience not only that there is no advantage in the maintenance of an even temperature, but that the natural daily fluctuations of temperature are of value to the patient, and I believe it would be a distinct step in advance if

[1] Die Deutsche Klinik, ii, 243.

we would discard from the sick-room the ordinary thermometer, and put in its place the wet and dry bulb thermometer, or one of its more readily interpreted and sufficiently accurate substitutes.

In tuberculosis the worst possible environment is a close, warm room, such as is maintained, as a rule, for pneumonia. Far better, but still inadequate, is a normally well-ventilated room, but by far the best is absolute out-of-doors. As a result of actual trial I have concluded that the same rule holds good for pneumonia.

In the case of adults, I have kept them in either a small room, or in the end of a ward, between two widely opened windows, with an absolutely free circulation of air, and at a temperature in winter that compelled the nurses to wear overcoats.

Dr. Northrup has carried the children with pneumonia to the roof in the early morning and kept them there throughout the day, and this in winter and often without even a wind-break.

I realize the difficulty of estimating the value of any plan of treatment in pneumonia from a study of the mortality statistics, that is, in a moderate number of cases; so I will merely state that the experiment was tried on my service at the Roosevelt Hospital and the Presbyterian Hospital, and in Dr. Northrup's service at the Presbyterian Hospital during the past winter, and the ultimate results, as far as mortality is concerned, were at least as good as with ordinary closed-ward treatment. More definite data can be obtained from a study of the symptoms, and we have noticed that exposure to the open air has had somewhat the same effect upon the nervous symptoms as has the cold bath in typhoid.

Nervousness and restlessness diminish, delirium is less frequent and less active, the patients sleep better at night, and the tongue is less likely to be dry.

I have not noticed any influence upon the duration of the disease, or upon the incidence of complications. I have been surprised to note that, as a rule, the patients have liked the treatment, and have not complained of the cold. In no case has the patient caught cold or developed a complication that could fairly be ascribed to exposure to a low or changing temperature.

Impressed with the satisfactory behavior of the nervous symptoms in pneumonia, I was encouraged to try the treatment of typhoid fever by the same environment.

The number of cases at my disposal was too small to admit of safe generalization, but as far as the experiment was carried the results were satisfactory, and suggested the likelihood that the fresh air treatment in this disease would be found in certain cases a satisfactory substitute for cold baths. The behavior of the tongue and of the nervous system suggested the conditions that are found when cold baths operate successfully. Even if of moderate benefit, though less so than baths, it would still be of much use in many

cases in which baths are contraindicated, or in which it is not possible to carry them out satisfactorily.

In private practice baths are of limited utility, and are not widely used, requiring so much and such skilled assistance as to be available only for the wealthy, while, unless carried out in the best manner, they are not free from danger. I suggest the substitution of fresh air treatment under those circumstances, which, in addition, has the advantage of harassing the patient less and subjecting him to no risk of injury.

The influence of fresh air upon the nervous system is easily demonstrable in many different diseases; but by fresh air I mean not what would be regarded as good ventilation in living-rooms, but fresh air as it is met with best out-of-doors, or, if this cannot be arranged, in rooms with wide-open windows. The evil effects of the opposite condition upon the nervous system are easily demonstrable. After a night spent in a room with closed windows and a temperature maintained by artificial heat, following wakefulness and restlessness through the night, there is apt to be headache, mental depression, a lack of buoyancy, a coated tongue, and a lack of appetite in the morning. These symptoms are not easily explained, but do not seem to be due primarily to deficient oxygenation, for they need not be present in persons who have diminished breathing space from disease of the lungs, and whose oxygenation is markedly below the normal. In several diseases in which disturbance of the nervous system plays a prominent part, these symptoms have diminished under the influence of a continuous stay in the fresh air. I have treated cases of typhoid fever lately by placing them before open windows, covered only by a sheet. The number of cases so tried has been too small for generalization, but the results are that such treatment may approximate in its effects upon the nervous system to those of cold baths, and the patients have found the procedure a pleasant one. One young woman with severe typhoid fever was kept in a room with wide-open windows, in which the temperature was at times 36° F. Along the line of Flügge's demonstration, I would suggest the use of fans systematically for these cases in warm weather. In no case have any unfavorable symptoms resulted from the treatment.

From many similar observations, I may quote two patients with cerebrospinal meningitis, who entered the hospital at about the same time, and occupied beds in the same ward, which was ventilated by the modern closed aspiration system and had the windows permanently closed. Both patients were severely ill with the ordinary symptoms of the disease. They were mildly delirious during the day, and were actively delirious during the night, and slept only when under the influence of opiates. Then the ventilating system was cut off from the ward, and the windows opened, the temperature falling to from 50° to 55° or even lower, and with a

very free circulation of air, the winter wind blowing freely through the room. At once the patients began to sleep better, and from this time no opiates were needed.

The same experience was met with in other cases of cerebrospinal meningitis, which was epidemic in New York in the winter of 1904 and 1905. Subsequently these cases were regularly segregated in special rooms where the fresh air method was carried out as an important element in their treatment.

In pneumonia it is especially the nervous symptoms, delirium, restlessness, etc., that are improved by out-door treatment.

Several years ago when the importance of this segregation and rational treatment of cases of tuberculosis in public institutions was first recognized, the late Dr. Dent, of the Metropolitan Hospital for the Insane on Ward's Island, removed the cases of tuberculosis in that institution to tents, and subjected them to modern, out-door treatment. He was prepared to see improvement in their pulmonary symptoms, but was surprised to find an equal or even more marked improvement in their nervous symptoms, especially a diminution in restlessness and violence, and a lessened need for restraint. He then tried the same environment upon the other inmates of the asylum, and found an equal improvement in their nervous symptoms; the number of the disturbed patients was less, and restraint was much less often called for. Some of the most successful sanatoria for the treatment of neurasthenia find absolute out-door life one of the most useful elements of treatment.

The influence of environment upon digestion is almost equally striking. By good or bad ventilation, by cheerfulness or gloom, by amusement or boredom, the appetite can be made vigorous or fickle, the tongue clean or coated, the digestion good or bad, and even the activity of the bowels may be modified. Nothing is more striking in places devoted to the out-door treatment of tuberculosis than to notice the enormous quantities of food that are taken with relish and digested with comfort by frail men and women whose lives are spent in steamer chairs on open piazzas without any activity, either bodily or mental. I have known patients with acute phthisis, when living thus constantly out-of-doors both day and night, maintain both appetite and digestion unimpaired until a few weeks before death. Experience surely suggests that it is the out-door element in the treatment chiefly that has kept the digestion thus vigorous.

In some of these very severe cases of nervous dyspepsia in which the usual remedies are of no avail, even when combined with rest in bed, I have known improvement begin when to the rest of the treatment was added fresh air out-of-doors; and I believe that this element is capable of being made of much use in combating this obstinate disease.

There are certain diseases in childhood in the management of

which environment can be made to play an important part, and in relation to which this form of treatment has been too much neglected. I saw a good example of these recently in conjunction with Northrup, to whom we owe many valuable and vivid suggestions in this direction. A boy of five, in good circumstances, had shown a mild type of rickets from an early age, in spite of the best feeding and suitable medication. Talking, walking, and dentition were delayed. There was a pot-belly and a slight rickety rosary, and the other symptoms of the disease were present, but in mild degree. Careful and judicious treatment, dietetic and medicinal, had failed to remove the trouble, although the child was living in the country in excellent surroundings. Then, at Northrup's suggestion, the entire scheme of life was changed. The parents were persuaded to build an open playground on the roof, and this the child occupied throughout the entire day, never going into the street nor to Central Park, although this was not more than a few hundred yards from his home. The bed-room windows were opened wide, and so thorough was the ventilation that a fur coat had to be provided for the trained nurse's use when she had occasion to get up in the night. In this way there was obtained for the child continuous life in the open air, free from dust contamination of the street, together with absolute freedom from nervous excitement and the stress of competition with the other children of the family—with a marked diminution in the number of visual, aural, and other mental impressions received.

The result was better even than had been expected, and though no new diet or medication was instituted, the child made more progress in the next two months than he had made in as many years. Here the treatment was entirely by environment, and I would suggest a wider application of the same principle in similar cases, and especially an effort to attempt its introduction in hospitals for children.

In the case above described, it was not entirely the open-air life, for he had passed the preceding two years in a healthful locality in the country, but it was the judicious combination of all the elements of environment that could be made of benefit to him.

In New York City the children's playground on the roof is becoming a common feature of many homes.

Last year during the summer in one of the Metropolitan Hospitals, in order to allow repairs to the wards, the children were removed to the yard and temporarily housed in tents. Not only was there marked and obvious improvement in the patients, but the commissary department of the institution drew attention to the bills for food, and complained that these were nearly doubled. There was some consolation, however, in noticing at the same time that there was a considerable lowering in the size of the bills for drugs.

The little hospital for non-pulmonary tuberculosis in children maintained by the Association for Improving the Condition of

the Poor at Coney Island, New York City, constitutes one of the best practical lessons in the use of environment. Here, the little patients spending the entire year on the seashore absolutely out-of-doors, or with unclosed windows, in all weathers, make remarkable progress, while colds and sore throats are unknown.

Several years ago in one of the Lying-in Hospitals of New York City, a patient with puerperal fever was put upon the roof, and kept there during the course of the disease. I have recently adopted the plan of putting these cases in an environment as nearly resembling out-of-doors as possible, and without regard to atmospheric temperature, combining this with as near perfect quiet as can be obtained in a hospital, and with forced feeding. The number of cases treated has not been large enough to justify a definite conclusion, but the results thus far certainly encourage a continuance of the experiment.

The influence of light upon disease is difficult to determine accurately. This agent is known to hinder the growth of bacteria and to aid in their destruction, and also to increase tissue oxidation. Curiously, the absence of a normal amount of light in the environment has been found by Weber to give rise to symptoms similar to those produced by a close atmosphere, namely, diminution of energy, depression of spirits with loss of appetite, indigestion, coated tongue. This field deserves further study. Sun baths or the exposure of the whole body to direct sunlight are used extensively in many sanatoria, and a valuable action upon the nutrition and upon the nervous system is claimed for them.

The action of *x*-rays, the Finsen light, radium, and other applications of light in the treatment of diseases indicate that this agent may be used with advantage in therapeutics, while Nevin's claims that epithelioma of the skin may be caused by the action of direct sunlight also suggests the potency of the light rays.

The exact part that light may be made to play in the management of environment in disease requires extensive observation and experiment to establish, but it is a field that would repay investigation. Meantime, consideration of the lighting and coloring of hospital wards and sick-rooms may certainly be made to play an important part in the therapeutic and the psychical side of environment.

But it is especially in connection with hospital practice that I desire to emphasize the importance of escaping from the trammels of tradition, and of starting anew to work out the treatment of the patients in part through environment, and of attempting to apply to the treatment of many diseases those principles that have proved of such inestimable value in a few, and which need not in any way interfere with such other therapeutics as may be indicated, whether specific or symptomatic. At present, as far as environment is concerned, the average hospital is an inelastic unit. Drugs may be varied to suit the needs of individual patients, and even the

diet to some extent, but the environment is rigid, and is adapted to but a small part of the inmates, if any.

In many institutions the profligate use of coal maintains the temperature of wards uniformly day and night at as near as may be to 70° F. A closed system of ventilation maintains an atmosphere that causes headaches in nurses and physicians who must spend many hours in the wards. The patients are assigned to this or that service, being distributed to suit the convenience of the visiting physicians. Hence, it comes that the sun shines alike upon the patient with acute tuberculosis, who needs it, and upon the one with cerebrospinal meningitis, who suffers the keenest photophobia. The bright, sunny ward, with raised curtain shades and walls painted a light color, is admirably adapted to the convalescent or the neurasthenic, but poorly adapted to the needs of patients with typhoid fever in the acute stage.

Almost more difficult to reconcile are the conflicting needs of the man with acute nephritis, general anasarca, and suppression of urine, and of the one with acute lobar pneumonia. But both, as a rule, must have the same atmospheric temperature, the same kind of ventilation.

I realize that the natural limitation of the function of the hospital precludes the possibility of providing separate rooms, or even small wards, for the patients. Moreover, the construction of most hospitals makes it difficult to provide for each individual the exact environment he would find best. Yet in most cases an intelligent effort to apply to hospital patients those principles of treatment by environment which are the outgrowth of modern scientific medicine, and some of which I have already briefly outlined, will undoubtedly result in making hospital results correspond more closely to those obtained in private practice.

In institutions with more than one ward devoted to each sex, these wards should be assigned to groups of patients according to their therapeutic needs, rather than to individual visiting physicians.

Thus, the wards could be made to vary in temperature, air currents, light, and quiet; and so some approximation at least of adjustment of surroundings to the needs of the sick could be carried out. The slight increase in administrative difficulty would be more than counterbalanced by added efficiency of service.

During the last year in two of the hospitals with which I am connected, I have attempted to carry out the above plan. In one institution I was allowed no expenditure of money whatever. The medical wards are the long, narrow type, containing twenty-eight beds each. At the far end is a hallway lying between the lavatories, and having a very large double window, and closed off from the wards by doors. This was used as an out-door space, the windows being widely opened and the door to the ward kept closed.

Here the temperature was not far above that of the street, and here were kept certain cases of pneumonia, or typhoid fever, or acute tuberculosis, within the limits of the space at my disposal.

One end of the ward itself was maintained with closed shutters and open windows, and here the air was fresh and the temperature varied with that out-of-doors, ranging from 45° to 60° or more. The closed shutters brought about a subdued light. In the other end the windows were closed and the shades raised.

I was surprised to find that the temperature in the two ends of the ward varied at times as much as four degrees, while the free circulation of air through the open windows made the apparent difference much greater.

Thus, without the expenditure of any money and with no structural changes whatever, I was able to secure in this ward three essentially different forms of environment, and kept the patients distributed in accordance with the nature and progress of their complaints.

In the other hospital, there had been certain superficial structural changes introduced, which made the conditions much more satisfactory. The ward had been partitioned and the wall of one part painted a dull green, and the windows provided with green shades. Here the windows are kept open, and acute cases, needing fresh air, quiet, and a subdued light can be given a suitable environment.

Meantime, suitable provision is being made on the hospital roof for the accommodation of all patients that would be benefited by absolute out-door surroundings.

I have been interested to notice that since the introduction of the above changes the health of the nurses has materially improved.

I would here put in a plea for the more general and systematic use of hospital roofs, and would suggest that the architectural definition of roof be changed, as far as it applies to hospitals, and that hereafter, this structure be defined as the floor of the top story.

The children's playground on the roof is rapidly becoming a familiar feature of the newer houses built in New York, while more and more hospitals are learning that their roof space can be made one of the most valuable therapeutic assets. With the downfall of tradition as the ruling spirit in the construction and utilization of hospitals, it might fairly be hoped that the assumption that medical hospital treatment is for the most part expectant might be done away with, and that our activities in behalf of our patients might rival even if they do not equal those of our confrères of the surgical side.

In the foregoing discussion it is not intended to try to cover more than a small portion of the environment in disease, but rather to

call to its consideration more general thought than, as a rule, it receives, and to suggest that the adjustment and maintenance of this element in therapeutics, both in private and in hospital practice, be reviewed in the light of the modern teachings of physics, physiology, and experimental medicine.

THE PHYSIOLOGICAL LIMITATIONS OF RECTAL FEEDING.[1]

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The title of the subject that has been given me to discuss implies, of course, a question to be answered; and the essence of that answer may be compressed into the statement that the limitations of rectal alimentation are very narrow—much more narrow, indeed, than is commonly appreciated. In saying this I mean only its limitations as a means of furnishing food, not, of course, its therapeutic limitations as a means of combating symptoms. There is a widespread feeling that when one is using rectal alimentation and the success of the measure is not interfered with by irritation of the bowel, one is using a substitute for natural feeding by the mouth that, while not perfect, is by no means a bad substitute. It is somewhat generally believed, indeed, that in a very considerable proportion of cases, the results are so good that the patients maintain a nutritive equilibrium or actually gain tissue, even though no food is taken in the normal way. This is not merely a common belief in the profession at large; it is the teaching of a large proportion of clinical instructors. As a general belief, however, it is extremely erroneous.

If one examines into the reasons for this prevailing opinion, one finds that it is partly due to the mere acceptance of tradition, partly founded upon expressed opinions of clinicians; these opinions, however, being in most instances the result of loosely gained impressions rather than of accurate observation. When patients so fed have been carefully observed, especially when they have been weighed, it is usually found that they lose in general nutrition and that they often lose a great deal of weight; and if the actual absorption is determined, the results seem, in almost all instances, still less encouraging.

Patients may, as is frequently claimed—and, indeed, they often do—feel better as the result of exclusive rectal alimentation than they did before; but this is by no means direct evidence that they have improved in nutrition. It is more commonly evidence that the local disease that is the cause of their chief complaints has improved.

[1] Read at the meeting of the Association of American Physicians, Washington, D. C., May 15 and 16, 1906.

So severe a measure is not ordered unless there is decided reason for it, such as protracted and troublesome vomiting, gastric ulcer, or obstruction to the passage of food through the œsophagus or the pylorus. If food produces vomiting or severe pain when taken into the stomach, stopping food by the mouth will usually cause great relief; but it does not wholly ease the mind of either the doctor or the patient, for neither of them can let absolute starvation go on for a considerable length of days without a disquieting feeling of danger.

Patients who are permitted no food by the mouth, and those that have, for instance, for a long time been more or less starved by œsophageal obstruction, have much of this disquiet and much of their sense of starvation dispelled, and the accompanying symptoms relieved, by the knowledge that they appear to be having a goodly amount of food administered in other ways than by the mouth. A considerable portion of whatever well-being is observed in patients that are nourished exclusively by the rectum is undoubtedly often due to this psychical effect. Such effects are by no means trivial and unimportant, and they are no longer scorned by those who would be scientifically accurate. Pawlow and those who have followed his suggestions have demonstrated in a highly illuminating way definite physical effects of psychical factors; and common experience shows the profound influence that such factors exert upon hunger and appetite. In the case of rectal alimentation, I know that the psychical effect can be very striking. In two cases in which Dr. Caspar Miller and I studied the absorption during exclusive rectal feeding, and in which, therefore, we knew just how much nutritive effect the enemas had, the patients told us that their hunger, which appeared intense, was quieted almost completely *within a few moments* after every enema; and each patient was quite convinced that she was being generously fed in this way; yet each was absorbing an amount so small that if the entire quantity obtained in twenty-four hours had been served at one meal to be taken in the usual way, it would have been a bitter disappointment to a hungry person; for the total absorption in each case in the twenty-four hours was just about equal in food value to a good-sized glass of milk.

Another reason why rectal alimentation actually does good without really furnishing much tissue-building food—a very important reason, and one that can be more clearly appreciated than the psychical effect—is because patients that require exclusive rectal alimentation are almost always suffering from lack of fluid as well as from lack of food. It is common knowledge that great reduction of fluid is more tormenting in its effects and more quickly disastrous than is reduction of food; and a moderate reduction, providing it is abnormal in its extent, differs only quantitatively in its effects from extreme reduction. Nutritive enemas always contain a large proportion of fluid; and most of this is quickly and readily

absorbed, and tends greatly to the well-being of the patient. It may, indeed, supply what is, for the time being, his most serious demand; and since a goodly proportion of the water may be retained by the parched tissues, the patient may even for a time gain in weight, and thus offer apparently important evidence that he is building tissue when he actually is not.

The salts, too, that are contained in nutritive enemas are in part absorbed, and they are undoubtedly of use in the organism and explain a certain portion of the useful effect.

In these ways, then, in furnishing mental satisfaction, water, and salts, rectal alimentation is always very useful when indicated; and it practically always provides in this manner a direct and positive gain for the patient. Were these purposes the only ones in view, exclusive rectal feeding might frequently be continued for a long time without harm, and often with profit.

The story is different, however, with the foods, as this term is commonly used; that is, with the organic tissue-building and energy producing elements—the protein, fats, and carbohydrates. All are absorbed to some extent when administered by the lower bowel; but all are absorbed much less freely than is commonly thought, and far less freely than when taken by the mouth. The total amount thus obtained is, in almost all cases, so small that, instead of benefiting the patient's nutrition by this means, one usually plays a losing game so long as it is continued. I would make it clearly understood that I do not mean that it is a harmful procedure, however. It may, in many cases, be knowingly and wisely undertaken. What I do mean is that, in a great majority of cases, the actual status of rectal alimentation as a method of nourishing patients is that it is a modified and partial starvation. It does furnish some food, and is, therefore, decidedly better than simple starvation; but it is nearly always a marked degree of starvation, and should be recognized as such and not be given a higher place than it deserves.

When used on account of troublesome vomiting or any other transitory cause, rectal alimentation is, of course, in suitably chosen cases, an entirely proper measure; and I would not offer the faintest protest against it. It is, however, important to recognize that, as a rule, one is by this means providing the patient with a wholly insufficient amount of nourishment; that it is, therefore, not to be undertaken lightly and with any feeling that nutrition is being well maintained; that more harm than good is likely to be done, if it is undertaken with the purpose, not merely of guiding the patient through a period of temporary severe gastric irritation or the like, but with the belief that it will improve general nutrition; and it must be remembered that, if other more promising measures can be used—especially if some surgical measure, such as gastrotomy for œsophageal stricture, is projected—one should not delay long

with rectal alimentation in the hope of making the patient's condition more suitable for operation. After sufficient water for the tissue-needs has been absorbed, the progress in general nutrition is usually downward, and the proper course to pursue is to operate soon; or in instances in which surgical measures are not in place—for example, when the trouble is prolonged severe gastric irritability—the proper course is to attempt soon to provide nourishment by the natural means, through painstaking choice of suitable diet and other measures. The length of time throughout which rectal feeding is in place is usually short. Ordinarily, to be sure, exclusive rectal alimentation is used for short periods only; but even then, the degree of starvation that it involves may be a matter of moment, and one should reckon with facts and not with traditions. When there is thought of using it for longer periods, this is still more important.

The foundation upon which I have based these statements is the work that has been done on the actual quantity that is absorbed from nutritive enemas. Together with Dr. Caspar Miller, I have made several such studies,[1] and in reporting them most of the important literature was discussed.[2] I need not, therefore, repeat at length the results of our work or that of others. These results demonstrate, in the main, that protein and fats show a very poor percentage of absorption by this channel; and when it is, at the same time, considered that the whole amount that can be successfully administered in this way is much below the amount normally taken by the mouth, the total actual absorption proves even smaller than the percentage-absorption would indicate. Some investigators consider that the fat-absorption scarcely passes ten grams a day; and, as a rule, the protein-absorption certainly but rarely equals twenty grams. Dr. Miller and I obtained slightly more favorable percentage-figures than many other workers; but, at best, we could reckon the value of all food absorbed in our most favorable cases at less than three hundred calories in a day, and usually it is not above two hundred calories. The latter amount is not one-sixth of the absolute demands of most persons, even of most sick persons, if they are to be kept from losing tissue, and especially if they are to have their nutrition improved.

Even these figures were, in our cases, and certainly in most others, unquestionably too high. In our work we made no attempt to determine the carbohydrate-absorption, for we considered the results too unreliable, owing to the disturbing factor of bacterial action. In order, however, to give as much credit as possible to nutritive enemas, we reckoned in all the carbohydrate as absorbed.

[1] University Medical Magazine, March, 1900; University Medical Bulletin, 1903; American Medicine, Feb. 4, 1905.

[2] Reach, Centralblatt f. die Grenzgebiete der Med. u. Chir., 1904, Band vii, Nos. 8 and 9, contains a very extensive collection of literature.

This we knew was erroneous, but we did not know how extensive the error actually was. In all probability, a very large part of the carbohydrate is really not absorbed. Investigators have varied in their opinion concerning this point, and in almost all the work, the extent of bacterial action could not be reckoned with exactly. The most valuable studies of the absorption of carbohydrates through the lower bowel that have been made are those of Reach,[1] who carried on his work by determining the effect upon the respiratory quotient, which is undoubtedly the most convincing indicator of the amount of carbohydrate that reaches the circulation. He obtained results that were very low, much below those that had been obtained merely by studying the amount remaining in the feces. If his results are correct, the figures that I have mentioned for the total nutritive value of ordinary rectal feeding are actually decidedly too high.

In a recent extensive study, Boyd and Macpherson[2] reached just about the same conclusions in regard to the protein and fats, as well as in regard to the total caloric value of this method of feeding, as those reached earlier by Dr. Miller and myself, and by most other recent students of this matter.

In regard to the carbohydrates, their views are more encouraging, but they are based upon the opinion that bacteria exert an extremely slight effect upon sugars in the lower bowel. Their experiments upon this point are, however, not entirely convincing; some important details are not mentioned, particularly the strength of the sugar-solutions that they inoculated with colon bacilli in order to study the effect of these bacilli in breaking up the sugar. In judging of the importance of their results, it is of considerable moment to know whether these solutions were dilute or strong. If the former, their results may be correct; but if the solutions were strong, they would hinder bacterial growth and activity. At any rate, studies of the same point by other persons have repeatedly indicated a very much more extensive action of the colon bacilli on sugars than that found by Boyd and Macpherson; and furthermore, in the intestine we are not dealing with colon bacilli alone, but with various other organisms that are capable of acting energetically upon sugars. At present the testimony certainly indicates that a very considerable part of the sugar administered per rectum is broken up by bacteria, and, therefore, is not received by the organism in a form suitable to maintain nutrition. That a noteworthy part of the protein and fats is also broken up and rendered useless, perhaps in part mildly toxic, is indicated by observations that Dr. Miller and I made on the ethereal sulphates; these studies as well as ordinary clinical observations show that intestinal putrefaction is often excessive when rectal alimentation is being used. Certainly, there is no

[1] Archiv. f. exper. Path. u. Pharm., 1902, Band xlvii.
[2] Scottish Medical and Surgical Journal, 1906.

reason to modify the statement that I have made that two hundred calories per diem is, with our present methods, a reasonable amount to expect most patients to absorb from nutritive enemas.

In rare instances, cases that have been carefully observed seem to have shown a larger degree of absorption than this. At times, indeed, practically the whole bulk of the enemas seems to have been absorbed throughout weeks of their use; and the patients have maintained good nutrition, or have even appeared to improve in nutrition. These cases are, I am sure, rare; and there are very few reliable instances of this kind on record. Personally, I have never seen such a case. Since, however, they undoubtedly do occasionally occur, an explanation for them is needed, if what I have previously said is correct. This explanation is, I believe, to be found, as I have stated elsewhere, in peculiarities in those persons that permit of the reflux passage of a large part of the nutritive enemas through the ileocecal valve. Digestion and absorption of the food would then occur in the small intestine, practically as in normal circumstances. This explanation is opposed to the views of a number of prominent students of digestion who have insisted that reflex peristalsis that carries large amounts of substances along the intestines does not occur. That, however, active reflex peristalsis does occur constantly in the large intestine has been very clearly shown by Cannon; and that in occasional instances the ileocecal valve does permit of the passage of large amounts of substances from the large intestine into the small intestine is clearly demonstrated by a number of cases that I referred to in previous papers,[1] by one that I observed myself,[2] by those cases mentioned by Rolleston,[3] and also by several cases reported from other sources. In these instances large quantities of easily recognizable substances introduced per rectum were carried backward throughout the whole course of the digestive tract, to be ejected ultimately per oram. Since this has been clearly shown to occur occasionally, I see no reason to question that it may be at least one cause of the occasional good absorption of nutritive enemas. An additional reason for believing this is the fact that most of the cases that have shown this reflux transport of substances from the rectum to the upper alimentary tract have been instances of hysteria, and likewise an extremely large proportion of the cases that seem to have been well nourished by rectal feeding were also cases of hysteria.

I may, in conclusion, refer very briefly to some possible ways in which rectal feeding may in the future be somewhat improved. The high food-value of fats (more than twice that of protein or carbohydrates) would make them extremely valuable for rectal

[1] Loc. cit.

[2] American Medicine, October 10, 1903.

[3] Editorial notes in American edition of Nothnagel's Diseases of the Intestines, p. 78.

feeding if they were moderately well absorbed, but, unfortunately, they are very badly absorbed. Their importance as food-stuffs led Dr. Miller and me to attempt to improve their absorption per rectum by trying to provide a form of soap that would be easily absorbed and not irritate the bowel; and also by trying to find means of emulsifying fats so that the emulsion would persist for a long time in the bowel, instead of being quickly destroyed, as is the case with the emulsion of fat in milk and egg-yolk, for instance. Our experiments with soaps were unsuccessful. Those with artificial emulsions were somewhat more encouraging, and we got rather better figures for absorption in this way than when we had used milk and egg-yolk. So far as we have gone, however, we have not advanced much beyond the ordinary methods of administering fats; and our process is, as yet, scarcely worth the additional trouble that it causes. It may be possible, in the future, to make some improvements that will largely increase the value of fats as a rectal food.

As to protein, von Noorden's suggestion (which he states that he is having investigated), that the ultimate digestion-products of protein (the various aminoacids, etc.) may be better absorbed by the lower bowel than native protein or albumoses and peptones, is worth attention, though serious difficulties, practical as well as technical, are almost sure to be met.

In regard to carbohydrates, it seems to me that the most useful suggestion is that of Reach, who found that dextrins had more effect upon the respiratory quotient than the other carbohydrates that he had studied, and appeared, therefore, to be better absorbed than others. On the basis of this, he recommends their use in nutritive enemas. They are, at any rate, usually soothing rather than irritating—contrary to the sugars—and they would also be much less easily broken up by bacteria than would sugars, perhaps scarcely at all.

One suggestion that has been made is that it may be possible, by introducing substances that tend to produce reversed peristalsis to encourage the passage of food from the large intestine through the ileocecal valve and thereby increase absorption in the small intestine, where it normally occurs readily. It is hardly to be expected that much success would result from attempts to carry this out; and indeed, from Cannon's observations of the normal occurrence of antiperistalsis in the large intestine, it would seem an irrational procedure, because unnecessary. The difficulty does not lie in the absence of movements in the large intestine that would carry the food up to the ileocecal valve, but in getting it to pass this valve. While I do believe, as I have stated, that in rare instances considerable amounts of food actually get by the valve, and that perhaps small amounts often or even constantly pass through, it is quite certain that, as a rule, no considerable portion succeeds in doing so.

No method of accomplishing this appears evident, and whatever may be done to improve rectal feeding, will, I think, be done in other ways.

THE RESULTS OF THE USE OF REFINED DIPHTHERIA ANTITOXIN, GIBSON'S "GLOBULIN PREPARATION," IN THE TREATMENT OF DIPHTHERIA.

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All who use diphtheria antitoxic serum extensively are aware that in from 10 to 30 per cent. of the injected cases of diphtheria, pronounced rashes, of an urticarial or erythematous type, occur. In the majority of cases these serum effects are disagreeable rather than harmful, but occasionally the rash is accompanied by constitutional disturbance presenting, in the most severe cases, high temperature, vomiting, prostration, and sometimes other symptoms. These marked constitutional reactions are especially likely to follow very large injections of from 10,000 to 20,000 units in young children who have high temperatures due to bronchopneumonia or other complications. In these cases the serum reaction is distinctly harmful, for by lowering the general resistance of the body to other infections it neutralizes to some extent the good done by the neutralization of the diphtheria toxin by the antitoxin. Furthermore, the rashes, especially those of a scarlatinal type, are puzzling in a diagnostic sense.

There have been many attempts made to separate diphtheria antitoxin from the non-antitoxic portions of the accompanying serum. Those interested in the chemical side of these investigations are referred to the recent article by Gibson.[1] In 1900 Atkinson, working in this laboratory, eliminated all but the globulins from the antitoxic serum. This partially refined antitoxic serum was tried in 36 cases. The results,[2] both as to antitoxic effect and serum reactions, were so nearly identical with those in an equal number of cases treated with the whole serum from the same horse that it did not seem to be worth while to go to the expense of preparing such an antitoxic solution. Attempts to effect a practical

[1] Journal of Biological Chemistry, vol. i, Nos. 2 and 3.

[2] Archives of Pediatrics, November, 1900.

separation of the antitoxin from a greater portion of the proteid non-antitoxic substances of the serum were continued. In August, 1905, we began trials with an antitoxic solution from which much more of the serum proteids had been eliminated than in the Atkinson preparation. Dr. R. B. Gibson, bacteriologist in the Research Laboratory, placed the half-saturation ammonium sulphate precipitate derived from the antitoxic serum in saturated sodium chloride solution, and found that along with a portion of the globulins all the antitoxin passed into solution. In this way the nucleoproteids and the insoluble globulins present in the Atkinson preparation were eliminated. The soluble globulins precipitated by acetic acid were filtered, partially dried, and finally placed in a sac of parchment membrane and dialyzed in running water. This antitoxic solution of soluble globulins was then rendered neutral, and sufficient sodium chloride was added to make it isotonic.

In carrying out the process there is a loss of about 30 per cent. of antitoxin units, because of retention upon filters, loss in dialyzing, etc. On testing this solution on a number of children we found that the results were favorable, except that more local pain was produced than with the whole serum. Stricter attention to the neutralization soon overcame this, so that when the serum was injected on one side and the globulin solution on the other the patient was unable to tell the one from the other. In October, 1905, the antitoxic globulin solution was administered by the medical inspectors not only in the hospitals for diphtheria but also in private homes. Since December it has been gradually distributed throughout New York City, and is now the only form of antitoxin supplied by the Health Department.

Results from the Use of Antitoxic Globulin Solution. The antitoxic effect was identical with that of the whole serum. Our tests have shown that not only the toxins and the so-called toxones produced in media by diphtheria bacilli, but also those produced in the animal by injections with living diphtheria bacilli are neutralized completely by the globulin solution. We could not detect the slightest evidence that any desirable substance in the antitoxic serum is lost by the refining process Not only we ourselves, but the resident and attending physicians watching the cases in the contagious disease hospitals noted that the rashes following the injections of the globulin solution seemed to be less severe than those which followed the injection of whole serum. It was especially noted that there were very few who had any constitutional disturbances even when the rashes did appear.

As the serum supplied by different horses, or from the same horse at different times, is known to vary in the rashes and other after-effects it produces, and as it is, therefore, difficult accurately to compare the globulin solution and the whole serum derived from different bleedings, it was decided to make a decisive test by collect-

TABLE I.—Results of Injecting the Mixed Antitoxic Horse Serum in Fifty Cases of Diphtheria Occurring in Children under Ten Years of Age.

Case.		Age in years.	Antitoxin units given.		Deleterious effects.	
					Constitutional disturbances.	Rash.
1	x	0.9	10,000	M	Marked, 5° rise of temperature.	Eighth day, general erythema lasting six days.
2		1	3,000	S	Slight	Tenth day, urticaria general.
3		1	14,000	M	High temperature, due partly to pneumonia.	Fourteenth day, urticaria lasting five days.
4	I	1.3	15,000	M	Moderate, 2° rise of temperature.	Eighth day, general erythema.
5	x	1.9	5,000	S	.....	
6	I	1.5	35,000	S	High temperature and marked disturbance.	Sixth day, urticaria; eighth day, general erythema of very severe type.
7	x	1 3	10,000	M	.....	
8	x	1.3	13,500	M	Moderate, with 1° rise of temperature.	Fourth day, severe general urticaria lasting three days.
9	x	1.6	10,000	M	Masked by pneumonia having 106° temperature.	Seventh day, severe general erythema.
10		1.7	10,000	M	Moderate, with 2° rise of temperature.	Sixth day, severe general erythema lasting three days.
11	x	1.9	10,000	M	Marked, with 3° rise of temperature.	Third day, morbilliform; eleventh day, severe general urticaria.
12		1.5	7,000	M	Slight.	Fourteenth day, erythema and urticaria general for two days.
13	x	1.5	10,000	S	Slight, with 1.5° rise of temperature.	Second day, quite severe erythema lasting one day.
14	x	2	17,000	M	Slight, with 1.5° rise of temperature.	Third day, very severe urticaria for two days.
15	x	2	10,000	M	Marked, but possibly due to sepsis.	Third day, urticaria and erythema very severe lasting fifteen days.
16		2	3,500	S	.....	
17	I	2.5	7,000	M	Moderate, 2° rise of temperature.	Fifth day, severe urticaria for six days.
18	x	2.5	14,000	M	Slight.	Thirteenth day, severe urticaria for three days.
19	x	2	3,000	S	Slight.	Twelfth day, general urticaria for two days.
20		2	10,000	M	Slight.	Sixth day, general urticaria for three days.
21		1.5	7,000	S	.....	
22		2.5	7,000	S	Extremely severe, 3°-6° for ten days.	Eighth day, morbilliform continued and intense for ten days.
23		2	7,000	S	Extremely severe, 2°-4° for one week.	Fifteenth day, morbilliform continued and intense for eight days.
24	x	2.5	8,000	S	Severe, 4° rise of temperature.	Tenth day, erythema for two days; seventeenth, second lasted six days.
25	x	2.5	7,000	M	Severe, 4° rise of temperature.	Seventh day, erythema for two days; twelfth day, second lasted five days.
26		2.8	12,500	M	Slight.	Twenty-second day, general erythema.
27	x	3	10,000	M	Severe, but possibly due to pneumonia.	Fourteenth day, erythema for five days.
28	x	3	10,000	M	.....	
29	I	3	8,000	M	None, except 1.50 rise of temperature.	Sixth day, urticaria for two days.
30	x	3	7,500	M	.....	
31	I	3	10,000	M	Severe, 5° rise of temperature for one week.	Thirteenth day, severe erythema for one week until death.
32	x	3	3,000	S	.....	
33	x	3	14,000	S	.....	
34		3	7,500	S	Severe, for ten days 4° rise of temperature.	Thirteenth day, severe erythema lasting ten days.
35		3	14,000	M	Very severe with 4°-7° rise of temperature for ten days.	Eighth day, general erythema over whole body for ten days.
36		4	3.750	S	.. ...	
37	x	4	10,000	M	.....	Twelfth day, severe erythema.
38		4	10,000	S	.....	
39	x	4	10,000	M	Moderate, 2° rise of temperature.	Sixth day, general erythema lasting three days.
40	x	4	5,000	S	.....	Fifth day, general erythema lasting three days.

TABLE I. (Continued.)

Case.		Age in years.	Antitoxin units given.	Deleterious effects.	
				Constitutional disturbances.	Rash.
41	x	4	10,000 M	Marked, 4°-6° rise of temperature.	Tenth day, very severe, lasting five days until death.
42		4.5	12,000 S	.....	
43		5	10,000 M	Moderate, 3° rise of temperature.	Fifth day, very severe, urticaria lasting five days.
44	x	6	5,000 S	.....	
45		6	10,000 M	Moderate, 2° rise of temperature.	Sixth day, general erythema lasting three days.
46		7.5	7,500 M	Marked, 4° rise of temperature.	Fifth day, general erythema. Eighth day, general urticaria.
47		9	10,000 S	Slight.	
48	x	8	3,750 S	.....	
49	x	8	3,750 S	.....	
50	x	9	3,750 S	.....	
50		3.24 years average.	Average units per case 9,250 } M = 28 S = 22	Thirty-five developed constitutional disturbances.	Thirty-six developed rashes.

x = intubated. I = croup. M = marked severity (of which eighteen were intubated).
S = slight severity. .... = absent.

ing a quantity of serum from four different horses, mixing it thoroughly, and then after precipitating one-half, to treat an equal number of patients simultaneously with the whole serum and with the globulin solution. These tests were carried out chiefly in the Willard Parker Hospital, but a few also in the Riverside Hospital. We are indebted to Drs. Lynah and Watson, the resident physicians in charge of these two hospitals, for their interest and aid.

It soon became evident that the serum we had chosen for the test was of such a character that eruptions and constitutional disturbances usually appeared in those injected with the whole serum. Whether it was because the serum from four long-treated horses had been mixed, or whether because of some other reason, it is certain that this serum produced more after-effects than any lot we had used in the hospital since 1899. These after-effects were so marked and occurred in such a large proportion of the children that we had to abandon the use of the whole serum. The rashes in those given the globulin preparation were much less severe. In persons over ten years of age almost no rashes occurred after either preparation. The patients treated with the whole serum and the antitoxic globulins were most carefully watched by us and the course of the disease, as well as the after-effects, noted.

After all the tested patients had become fully convalescent or had left the hospital, the histories were finally gone over and compared. It was found that fifty children under ten years of age treated with the whole serum had lived at least nine days, or long enough for the development of serum effects. The first fifty consecutive cases

TABLE II.—Results of Injecting Refined Antitoxin (Antitoxic Globulins) Made from Serum Obtained from the same Horses and at the same Bleedings as the Antitoxic Serum Used in the Cases Given in Table I.

No.	Age.		No. units of antitoxin. Severity.		Deleterious effects due to antitoxtin.	
					Constitutional disturbances.	Rash.
1	0.5		7,000	S	.....	Sixth day, moderate urticaria and erythema lasting four days.
2	0.9	X	15,000	M	.....	Second day, general erythema lasting two days.
3	1	I	10,000	M	Masked by pneumonia.	Ninth day, general erythema lasting five days until death.
4	1.5		5,000	S	.....	
5	1.5	X	12,000	M	Rise of 1° of temperature.	Third day, urticaria for one day.
6	0.3	X	7,000	S	.....	Second day, general erythema for three days
7	1.5		12,000	M	.....	
8	.4		3,000	S	.....	
9	1.2	X	10,000	M	.....	Tenth day, urticaria lasting four days
10	1.2		15,000	M	.....	Eighth day, urticaria lasting two days.
11	1.5	X	12,000	M	.....	Eighth day, urticaria, pretty severe, lasting three days.
12	1.3	X	12,000	M	.....	
13	0.9		5,000	M	.....	Fourth day, erythema lasting thirty-six hours.
14	.5		7,000	M	.....	
15	1.5	X	12,000	M	.....	Fifth day, urticaria for one day.
16	2		10,000	M	.....	
17	2	I	12,000	M	.....	
18	2	I	10,000	M	.....	Seventh day, mild urticaria for one day.
19	2	I	12,000	S	.....	
20	2		24,000	M	.....	
21	2	I	7,000	S	.....	
22	2		7,000	S	.....	
23	2	X	10,000	S	Masked by pneumonia.	Thirteenth day, general erythema lasting three days.
24	2		10,000	M	.....	Tenth day, general erythema lasting three days.
25	2.5	X	12,000	M	Rise of 1° of temperature.	Fifth day, urticaria, then erythema —together lasting five days.
26	2 5	X	12,000	M	Rise of 3° of temperature for twelve hours, then normal.	Seventh day, urticaria for two days.
27	3	X	17,000	M	.....	
28	3	X	10,000	M	.....	Eleventh day, erythema for two days.
29	3	I	12,000	M	.....	[days.
30	3	X	20,000	M	.....	Sixth day, urticaria lasting two
31	3	X	12,000	M	.....	Second day, slight general erythema lasting twenty-four hours.
32	4		7,000	S	.....	Sixth day, urticaria lasting two days.
33	4.5	X	8,000	M	.....	
34	4	I	12,000	S	.....	
35	4.5	I	12,000	M	.....	
36	4	X	12,000	M	.....	
37	4		12,000	M	Rise of 2° of temperature for one day.	Eighth day, severe urticaria, traces lasting five days.
38	4		12,000	S	.....	
39	5		5,000	S	.....	
40	5		12,000	M	.....	
41	5		3,000	S	.....	
42	5		19,000	M	Rise of 2° of temperature for one day.	Sixth day, severe erythema, traces lasting seven days.
43	5 5		3,000	S	.....	
44	6		10,000	S	.....	
45	6		3,000	S	.....	Sixth day, urticaria and erythema for three days.
46	6		4,000	S	.....	Seventh day, urticaria for two days.
47	7	X	12,000	M	.....	
48	8.5	X	24,000	M	.....	
49	9		7,000	S	.....	
50	9		3,500	S	.....	
50	3.18 years average.		10,600 units average injections.	M = 31 S = 19	Constitutional disturbances, 5; possibly 7.	Rashes in 23.

X = intubated. I = croup. M = marked severity. S = slight severity. . . . . = absent. Of the fifty there were thirty-one of marked severity; eighteen of these were intubated.

TABLE III.—Comparative Table Giving a Summary of the Constitutional and Local Reactions Obtained in the Treatment of Fifty Cases of Diphtheria in Young Children with a Lot of Antitoxic Serum Derived from Four Horses and of an Equal Number of Similar Cases Treated with a Solution of the Antitoxic Globulins Derived from a Portion of the Same Lot of Serum.

	Children who were treated with the whole serum.	Children who were treated with the antitoxic globulins.
Marked constitutional symptoms accompanied by severe and persistent rash in	28 per cent.	0 per cent.
Moderate constitutional symptoms accompanied by a well-developed erythema or urticaria	18 "	4 "
Very slight constitutional disturbance accompanied by a more or less general rash	20 "	8 "
No appreciable constitutional disturbance, but a more or less general urticaria or erythema.	4 "	34 "
No appreciable after-effects whatever	30 "	54 "

TABLE IV.—Duration of Rashes.

	Days.								
	1	2	3	4	5	6	7	8 and over.	Total rashes.
Antitoxic globulin cases	4	7	5	3	3		2	...	23
Whole serum cases	1	4	10	1	10	3	2	5	36

in children under ten years treated with the antitoxic globulins precipitated from the same lot of serum and living nine days or over were taken to compare with these. Table I gives the salient points for each case treated with the whole serum and Table II the same for those treated with the solution of antitoxic globulins. Tables III and IV summarize these points.

It is noticeable that not only were the rashes more frequent, but also much more persistent in the patients who received the whole serum. Twenty-three rashes following the use of the whole serum lasted over three days in this series, as against only six in the antitoxic globulin cases.

SUMMARY AND CONCLUSIONS. The results obtained in these series of one hundred cases are so definite that it seems safe to conclude that the removal of a considerable portion of the non-antitoxic globulins, as well as the albumins from the serum by the Gibson method, has eliminated much of the deleterious matter from the serum, so that severe rashes, joint complications, fever, and other constitutional disturbances are less likely to occur from the antitoxic globulins than from the antitoxic serum from which it was obtained. The globulin preparation when tested by animal experiments appears to retain all the antitoxic properties of the whole serum. The portion of the globulins still accompanying the antitoxin in the Gibson preparation is shown to be capable of exciting rashes and

occasionally constitutional disturbances, although, as stated above, to a less extent than the serum. It is almost certain that methods will be devised to refine antitoxin still farther, and so possibly eliminate all appreciable deleterious effects of the antitoxic serum.

Whether this globulin solution will be much less likely than the serum to cause collapse in the rare cases of peculiar susceptibility, such as in a certain percentage of those suffering from status lymphaticus, is still undetermined. It has now been used in several thousand cases of diphtheria without accident.

The concentration of antitoxin made possible by the elimination of the non-antitoxic substances is not only a convenience, but of distinct clinical importance, as it tends to encourage large doses.

The antitoxic globulin solution, like the serum, tends to become slightly cloudy when kept at moderate or high temperature, and substances such as solutions of carbolic acid and trikresol are especially likely to cause a precipitate to develop. The antitoxin in the globulin preparation retains its potency about as long as that in the whole serum.

THE RELATION OF HODGKIN'S DISEASE TO LYMPHOSARCOMA.

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Great confusion has existed since enlarged lymph nodes were first studied, as to the exact relationship between Hodgkin's disease, leukemia, malignant neoplasms, tuberculosis, and syphilis. Hence, a variety of names have been introduced into literature, implying a relationship to one or the other of these, which have led only to more confusion, two authors describing the same condition under different names or different conditions under the same names. Therefore, we have lymphoma (Wunderlich[1] and Ranvier), Hodgkin's disease (Wilks[2]), lymphosarcoma (sarcoma lymphomatodes) (Virchow[3]), malignant lymphoma (Billroth[4]), pseudoleukemia (Cohnheim[5]), and adénie (Trousseau[6] and other French authors).

In order to make clear the terms used in this paper I shall define them according to the most generally accepted views at the present time. By Hodgkin's disease is meant a constitutional disorder

[1] Archiv. f. Physiol. Heilk., 1858, Band xvii, p. 123; also Archiv. f. Heilk., 1866, p. 531.
[2] Guy's Hosp. Rep., 1865, xi, p. 56.
[3] Die krankhaften Geschwülste, Berlin, 1864, Band ii, p. 728.
[4] Gen. Surg. Path. and Therap., 1869 (translation of fourth German edition).
[5] Virchow's Archiv., 1865, Band xxxiii, p. 451.
[6] Clinique Med., 1865, tome iii, p. 555.

accompanied by enlargement of the lymph glands and the lymph-adenoid tissue in various organs, which enlargements remain discrete, with clinical manifestations of great increase in size of all or some of the lymph glands, enlargement of the spleen, fever of uncertain type, marked secondary anemia, no leukocytosis, and severe cachexia, resulting in death. By lymphosarcoma is meant a growth originating in lymphoid tissue which may or may not be accompanied by enlargement of other lymph glands, which does not remain confined to the lymphoid tissue, and which infiltrates and destroys surrounding tissue.

My object in this study has been to endeavor to determine the relationship between Hodgkin's disease, as defined by Dorothy Reed[1] and seconded by Longcope[2] and Simmons,[3] and malignant growths. I have studied nine cases, six of which correspond in most respects to the cases of these observers; while three, although showing the same clinical picture and the exact histological structure as did their cases and as do the other six of my cases, show also an undoubted malignant character, by affecting not alone lymphoid tissue, but infiltrating and destroying adjacent structures.

The disease occurs more frequently in males than in females. Of my nine cases one was in a boy of eleven years, one in a man of sixty-five, while the others were in men between thirty-three and forty. An exciting causative agent has never been conclusively demonstrated. Many bacteria have been assigned this duty, but there has been such a variety isolated from the lesions as to preclude the idea that any one is the specific cause. Fischer[4] in twelve cases, found cultures from the blood and glands sterile.

For the last twenty years many authors have advocated the view that Hodgkin's disease is a peculiar condition due to infection with the tubercle bacillus. Delafield,[5] in 1887, Weishaupt,[6] and others described cases simulating tuberculosis. Sternberg[7] in 1898, reported thirteen cases in which eight showed signs of tuberculosis, but the careful work of Fischer, in 1897, and of Reed, Longcope, and Simmons, in the last two years, has practically proved that tuberculosis and Hodgkin's disease are separate entities, though the one may be superimposed upon the other.

Benda,[8] in 1904, advocated the view that the disease is not produced by a specific infectious agent, but by modified or attenuated toxins of several infectious agents.

[1] Johns Hopkins Hospital Reports, vol. x, p. 133.
[2] Bulletin of Ayer Clinical Laboratory, October, 1903, No. 1.
[3] Jour. Medical Research, 1903, iv, 378.
[4] Arch. f. klin. Chir., 1897, Band lv, p. 467.
[5] Medical Record, 1887, I, 424.
[6] Pseudoleuk. u. Tuberculose, 1891, Inaug. Dissert., Tübingen.
[7] Zeitschrift f. Heilkunde, 1898, Band xix, p. 21, and Centralblatt f. d. Grenzgebiete d. Med. u. Chir., 1899, Band ii, pp. 641, 711, 770, 813, and 847. Lubarsch and Ostertag. Ergebnisse, 1903, ix, 302.
[8] Transact. Deut. Path. Gesellschaft, 1904, vii, p. 123.

Of my nine cases, six came to autopsy, and I had opportunity to study the organs of five; of the others, portions of the tumors only. Of the five, all showed an involvement of lymph-glands all over the body, with metastases in the liver in four, in the spleen in four, in the kidney in two, in the lungs, pericardium, pancreas, in one, each. Of the other four, one showed involvement of all the external glands and of the retroperitoneal; two of the cervicals and axillary; and one of the cervicals, supraclavicular, and mental. In five, the spleen was markedly enlarged, in one it was not palpable, in four it was not recorded. In five the glands were discrete, elastic, and easily movable; in one the cervicals were firmly attached to the fascia. In one was a tumor originating in the thymus gland and spreading by direct involvement to the pericardium and left lung, with metastases in the lymph glands, lower lobe of the lung, spleen, and liver. In two there was a large adherent tumor mass of the neck which involved the surrounding structures, fascia and muscles; and in one, part of a cervical vertebra and the wall of the jugular vein which passed through the tumor. In all of these, both in the infiltrating tumors, lymph glands, and visceral metastases, the macroscopic and microscopic appearance was practically identical, the variations being due to difference in the stage of development or rate of growth. In six of the cases the first sign of enlargement appeared on one side of the neck, in one, on both sides simultaneously, while in two it was not stated. In four of the cases the enlarged glands were removed by operation, and in all there was a recurrence after a few weeks.

The duration of the disease is agreed to be usually from three months to three years, though one of Dr. Reed's patients lived seven and a half years. One of my patients lived three and a half, two over one and a half years, but all the rest died in from three to ten months after the first symptoms appeared.

The disease runs a fairly typical course to the end, which is always death. It begins by an enlargement of one or two sets of lymph nodes, most often the cervical, which gradually extends by continuity to involve successively those of the whole body. In some cases only one or two sets may be affected, as in a case of Longcope's, in which the retroperitoneal and bronchial sets alone were involved, and in one of my cases (IX), in which only the cervicals were enlarged. The spleen is usually enlarged. There is seldom any pain in the swellings themselves, but the symptoms are often produced by pressure of the tumor masses, as in Case IX, in which the tumor pressing on the pharynx caused difficulty in breathing and swallowing, and œdema of the face and neck, and in Cases I and IV, in which there was œdema of the lower extremities from pressure on the vessels.

When the glands begin to enlarge the patient usually notices no impairment in health; but as the growths progress, there is a

more or less rapid loss in weight and in strength, an increasing cachexia, and secondary anemia. The blood picture is normal at first, later showing a gradually increasing secondary anemia, with no relative change in the leukocytes (Case I). Sometimes there may be a transient leukocytosis or a terminal increase, but these can be explained by intercurrent infections, as in Case VIII. In our cases the blood of six showed practically normal leukocyte counts, with growing anemia in one and more or less secondary anemia in the others.

Moderate fever is often noticed. It may be slight and constant or marked and irregular. A good many cases have been reported in which it showed a markedly remittent character, resembling that of remittent malarial fever or Malta fever—as occurred in one of our cases (Case I). This at one time was thought to be characteristic of the disease, but so much variance has been noted in different cases that no certain character can be assigned to the fever.

The diagnosis is easily made from true leukemia by the absence of the excessive number of leukocytes peculiar to that disease, with the exception of those cases of leukemia in which there is no marked leukocytosis. (In such instances a differential count would probably clear up the matter; see Blumer.[1]) From tuberculous adenitis the diagnosis is not so easy. In advanced cases the picture is typical, but when the glands are just beginning to enlarge it is almost impossible to separate the two clinically in some instances. If a gland be removed and examined microscopically the difference is easily made out.

THE TUMORS. The lymph glands in all my cases showed much the same appearance and corresponded closely to those described by most writers. They were usually round or bean-shaped, with definite capsule, and in the large masses remained for the most part separate, though the glands were frequently connected by pedicles from the pulp. In two of the cases, however, in which the large infiltrating tumors of the neck existed, while such discrete glands were present in the periphery of the tumor mass, the growth itself seemed to be composed of a large number of glands which had coalesced. Through it ran thick bands of connective tissue, probably the remains of the old capsules. The surface presented an irregular nodular appearance, as of glands with capsules on the outside, which had fused together on the other side. No capsule could be distinguished in places, and here the tumor infiltrated and destroyed the fascia, sternomastoid and other muscles, and the salivary glands. In one case the tumor mass extending down through the upper aperture of the thorax surrounded and infiltrated the wall of the jugular vein. In the case in which the tumor originated in the thymus gland it showed a coarsely lobulated surface covered with a smooth, fibrous capsule; though where it infiltrated the lung and pericardium there was no capsule, but the nodules were surrounded directly

[1] Loc. cit.

by the tissues of the organs. The enlarged cervical, mediastinal, and retroperitoneal glands, and those about the pancreas and spleen presented the same appearance as the glands in the other cases.

The lymph glands vary in size from 1 or 2 mm. to 5 or 6 cm., and vary greatly in consistency. In three of my cases (II, III, and IV) they were extremely soft and elastic, yet solid, having shown clinically a rapid growth. In other cases they were quite hard and firm (Cases I and V), having shown clinically a more chronic course. In Case IX the tumor showed necroses and liquefaction in the centre, but the glandular masses about it, though extremely soft, were solid.

On cut surface all the glands and tumors showed the same general character—a prevailing grayish white, with yellowish bands running irregularly from the capsule so as to cut the surface unevenly. These bands were harder than the grayish tissue between them which protruded above. In the softer glands (Case IV) the surface was uniform, showing no bands, and was a very light, moist, glistening gray, protruding far beyond the capsule. In the harder ones the surface was less moist and of a dull color, while whitish areas protruded beyond the bands.

In some of the glands (Cases IV and IX) small yellowish opaque areas of necrosis appeared, and in several small hemorrhagic areas were seen (Cases IV and IX).

The metastases in the various organs presented similar appearances. They varied from pin-point masses to those 1 cm. in diameter. They were sharply outlined, without capsule, and on the cut surface similar to that of the lymph glands. They were very irregular in contour. In the liver, lying in the periportal connective tissue, they grew irregularly therefrom. In the spleen they seemed to originate in the Malpighian bodies.

Microscopic Examination. In all the cases the growths showed some stage of the same process, and corresponded closely with those recently studied by Reed, Longcope, and Simmons. In the early stages a proliferation of the cellular elements takes place rapidly. The histological structure of the gland may still be apparent, but the follicles are enlarged and the lymph sinuses plugged with cells. There are evidently three sources of this proliferation: (1) From the germinal centres of the follicles, (2) from the endothelium lining the sinuses; and (3) from the fibrous tissue of the reticulum, capsule, and bloodvessels. The lymphocytes are mostly of the variety found at the centres of normal follicles, with a somewhat larger and less densely staining nucleus than the ordinary variety. The endothelial cells in proliferating form large masses of cells of epithelioid character completely filling and distending the sinuses. Many of these show karyokinetic figures. Among these cells are seen large ones of the same character, with one, and often with several large, clear, vesicular nuclei, arrayed at the centre

or unequally distributed in the rather dense, finely granular protoplasm. Reed has traced the development of these giant cells from the proliferating endothelium; and, from their position and appearance, my work would seem to confirm this view. The reticulum gradually becomes increased in amount and thickness, but only in the later stages is it marked. In this early stage there are many fibroblasts derived from the connective tissue. Eosinophiles are often seen in large numbers and occasionally a polymorphonuclear leukocyte and a plasma cell.

The proliferation may go on simultaneously from the three sources or one may outstrip the others. In two of the cases (III) and IV which showed rapid growth, the lymphocytes of recent type predominate and only here and there can an epithelioid cell or a giant cell be seen; whereas in another case (Case II) of even more rapid development, the epithelioid cells show most marked proliferation, and the sinuses appear as great light bands between the lymphoid nodules, in which no cell division can be distinguished among the large oval clear nuclei. The numerous mytotic figures are evidence of the rapid nuclear proliferation. These areas of syncytium have been described by Weishaupt,[1] and observed by Longcope in one of his cases.

These changes represent the soft variety of Hodgkin's disease; but, as the process continues, the growth of connective tissue takes place at the expense of the other cells. The reticulum is increased so that the cells seem held in a fibrous meshwork, while the large bands of connective tissue growing from the capsule, the old trabeculæ or the bloodvessels, traverse the pulp in various directions, dividing the gland into lobules of unequal size—which, as the disease advances, may occupy more area than the cells which are crowded together in little islets. From the proliferating reticular cells arise a variety of giant cells other than the ones described, which have six to ten small, rather deeply stained, oval nuclei arranged peripherally in the cells. Virchow first described these and Ribbert[2] traced their origin to the fusion of connective tissue and endothelial cells. He demonstrated their formation in experimental inflammation of the lymph glands in guinea pigs. They occur quite frequently in my cases and resemble in some respects Langhans' giant cells of tuberculosis and syphilis, but are easily distinguished from them. The structure of the normal gland is entirely lost in this later stage and the cells bear no particular relation to previous position of follicles or sinuses. The lymphocytes of the ordinary small variety are most numerous, many large epithelioid cells and characteristic giant cells are present, and in some cases numerous eosinophiles and some polymorphonuclear cells, but all of these

[1] Arbt. aus dem Gebiete der path. Anat. u. Bact. aus dem path. Anat. Inta. z. Tübingen, 1891 and 1892, Band i, p. 194.

[2] Ziegler's Beiträge, 1890, Band, vi, p. 187.

are scattered irregularly through the rather dense reticulum. Dr. Reed states that the capsule of the glands usually shows no change, except possibly some thickening. To this, from the study of my cases, I cannot agree. The capsules in all of my cases show infiltration, first with a few lymphocytes in a row between bands of the widened capsule, then as nodules with the typical histological picture of the disease. In three of my cases (II, III, and IV) I saw the tumor mass breaking through the capsule and forming a large mass on the outside, which in turn was walled off by a secondary capsule. So that the capsule of the large gland as we see it now is by no means the original one stretched out, but a composite capsule formed by secondary envelopes which have grown to wall off outgrowths from the gland which have destroyed and broken through the original capsule. This explains the thick bands which divide so many of the glands into flat concentric sections, for many of these bands are seen to be in direct continuity with the original capsule. And it explains, I think, the small collections of cells outside the capsule which Longcope took for a regenerative process by which new glands were formed to assume the function lost by the disease glands, which were in their turn only to be attacked by the process. Probably in some part of his specimen he would find that these small collections had a connection through the capsule with the already diseased gland.

In a lymph gland of Case II this was shown very beautifully. The gland was one of recent change, follicles and lymph spaces could be made out, the capsule was much thickened, and the lower layers much infiltrated with tumor; while, in one place, a stalk of the tumor extended through the capsule connecting with a small tumor mass outside which had a thin connective tissue envelop united at both sides with the original capsule of the gland.

In one of the cases (IX) in which the nodes in the centre of a mass of enlarged glands of the neck were fused together into a soft nodular tumor, a section showed the direct infiltration of a muscle, and the cells were pushing their way into and between the muscle cells, destroying them. In another place this tumor was seen surrounding and growing into the submaxillary salivary gland.

In the interior of thick connective tissue bands the tissue often showed a hyaline change (Cases V and VI); often connective-tissue cells and cells in the centres of cellular areas showed fatty change (Case V); and necroses were quite common.

The metastases in the viscera always presented the same histo-ogical picture as the lymph glands. They evidently begin as small collections of lymphocytes and proliferating connective tissue-cells which rapidly increase and assume the characteristic structure. They seem to arise in the vicinity of bloodvessels, as in the liver, the smallest ones are in the periportal connective tissue, spreading from there so as to coalesce into large irregular masses. In three

of my cases with metastases in the liver and general enlargement of the lymph glands the tumor was seen to be infiltrating the wall of a vein and protruding into its lumen (Cases I, II, and III). The boundaries of the growths are usually rather sharply defined, but without capsule; in places they can be seen growing between the liver cells, destroying them.

In the spleen the nodes appear more frequently in the Malpighian bodies and stretch out in ill-defined nodules into the surrounding pulp. The whole spleen shows an increase in the reticulum and a hyperemia; necrotic infarctions are quite common.

In the case in which the lung was affected, metastases showed plainly that they were not alone developments of pre-existing lymphoid tissue in the organ, but progressed as true malignant metastases by pushing the tumor structure already formed into adjacent tissue, destroying them, and occupying their places, while large nodular metastases occurred in the lower lobe removed from the invading part of the tumor and not in the vicinity of the bronchi—that is, in a situation where no lymphoid tissue exists normally.

Discussion on the Nature of the Process. Two possibilities as to the nature of the process are apparent, viz., is it a malignant, or is it an infectious process of the character of a granuloma? Most recent writers are inclined to the latter view. Reed is most insistant. Fischer, Clark,[1] and Simmons also hold this view. Longcope seconds it, though guardedly. Recent German publications also favor this view. Yamasaki,[2] regards the condition as a granuloma not of tuberculous but of unknown origin, which, however, may end in sarcoma. Wamecke,[3] although he has clearly recognized that lymphosarcoma cannot be separated from Hodgkin's disease, still adheres to this conception. From the study of my cases I incline strongly to the malignant theory. The points leading these authors to accept the infectious theory may be listed as follows:

1. The clinical picture. The disease may run an acute or a chronic course, accompanied by fever.
2. The frequency with which the disease starts in the cervical glands, suggesting an infection from the throat or skin.
3. It may remain quiet, then break out suddenly and cause death.
4. One tissue, the lymphoid, only is affected.
5. The mode of spread from affected glands to those nearest it.
6. Final stage of cachexia, diarrhœa, hemorrhages, etc.
7. Beneficial effects of arsenic in the treatment.
8. Histological appearance of the growth resembles that of an infectious process.

[1] British Medical Journal, 1901, ii, p. 701.
[2] Zeitschr f. Heilk., 1904, p. 269.
[3] Mitt. aus den Grenzgeb., 1905, xiv, 275.

9. The lack of infiltration of the capsule and surrounding tissues.

10. Metastases are caused not by cellular transplantation, but by proliferation of pre-existing lymphoid tissue apparently anywhere in the body.

It will be seen at once that many of these facts belong to malignant tumors as well as to infectious processes. We may have a rapidly growing sarcoma or we may have a slow one. We may have it accompanied by fever or we may not; and the fact that the fever of Hodgkin's disease is so variable in character, and sometimes not present at all, rather argues that it is not one produced by a definite infection. We see sarcomas which have existed for a long time, suddenly assume a very much accelerated growth. The mode of spread from diseased glands to adjacent ones is equally characteristic of malignant growths and of infectious diseases. The final stage of anemia, cachexia, and disturbances of the body functions is very characteristic of the last stages of all malignant growths.

That arsenic benefits the sufferers of the disease to any great extent is questionable; it never has cured. Everyone recognizes that there are periods of quiescence in the disease and even periods when the glands grow smaller, and this even when arsenic is not administered. The experiences of Köbner[1] and Lassar also show that arsenic has at times a decidedly beneficial influence in cases of malignant tumors.

That the metastases always start by a growth in pre-existing lymphoid tissue in the organs is not conclusively proved. In one of my cases it certainly was not the case, for a direct involvement took place from the tumor in the lymphoid tissue through the pleura and into the lung, with no regard to the situation of lymphoid tissue.

That infiltration of the capsule of the lymph glands occurred in all my cases, that in five cases the tumor showed undoubted extension through the capsule, and that in two cases involvement of muscle, salivary glands, and fascia occurred are clear evidences of malignancy. The recurrence after being removed in four cases is another suggestive fact.

In the liver of three of the cases (Cases I, II, and III) the tumor was observed invading the walls of a vein and passing through it. This was also noted once in the jugular vein. Reed observed this in the liver of one of her cases, and Longcope in the spleen of one of his.

Three of the cases were undoubtedly malignant, infiltrating and destroying surrounding structures as does any malignant growth. The only question that remains is, Can these be classed with

[1] Berl. klin. Woch., 1883.

Hodgkin's disease, or are they different and to be classed as lymphosarcoma? Osler[1] asserts that infiltration of the lung, as took place in one of my cases, does not occur in Hodgkin's disease, and that when such an infiltration does take place the disease is true lymphosarcoma. But in the light of the fact that this case, as well as the two other malignant tumors of the neck, present the same clinical features and the exact histological picture as the twenty-three cases so carefully studied by Reed, Longcope, and Simmons, and as the other six of my series, they must also be regarded as the same morbid condition. This being established, there would be no question as to the malignant character of Hodgkin's disease.

It is asserted by those who hold the infectious theory that this typical histological structure has more the appearance of an inflammatory change than that of a malignant growth. This appears to me not to be the case. The tumors resemble markedly in many respects true sarcomas. The irregularity of arrangement; the connective-tissue stroma, which so often occurs in other sarcomas, and similarly bears a very intimate relation to the cellular elements, forming a heavier connective tissue framework and a more delicate reticulum of thin fibers, which run between the individual cells; the large number of mitotic figures, evidence of a rapid proliferation of the cells, which are developed at the site and not in a great measure attracted there; and the peculiar forms of giant cells which bear more resemblance to those of sarcoma than to any found in inflammatory processes, all present a picture very suggestive of malignant growth, quite distinct from the different forms of infectious granulomas.

In suggesting a name for the disease I would agree with the recent German writers and preserve the term Hodgkin's disease.

I should reject pseudoleukemia as implying a relation to leukemia which is by no means proved. Lymphoma is too broad a term, for there are several forms of lymphoma. Malignant lymphoma and multiple sarcoma, implying a malignant character, although I believe it, cannot be accepted until more work has been done upon the subject.

I should reserve aleukemic lymphoma for a form of lymphoid tumors resembling those of true leukemia, but without the blood picture of that disease; these have been described by Blumer,[2] and others.

Six of my cases were clinically Hodgkin's disease, two were clinically malignant tumors, while one was on the border-line between the two. But as the lesions of all showed the same histological structure, I regard them as phases of the same morbid process. However, in view of the fact that clinically there is considerable difference between the extreme types it might still be advisable

[1] Quoted by Hare, Practice of Medicine, 1905, p. 775.
[2] Albany Medical Annals, April, 1905.

to retain for them the usual names: Hodgkin's disease and lymphosarcoma, respectively; always bearing in mind, however, that histologically there is no difference and that they are connected by transitional forms.

In conclusion:

1. I agree with Reed, Longcope, and Simmons as to the histological picture presented by the tissues in Hodgkin's disease, but I do not agree that it is necessarily due to an inflammatory process.

2. I assert that, in most cases, infiltration of the capsule of the diseased glands can be observed; in many cases an extension beyond the capsule occurs, and in some cases very evident infiltration of adjacent structures.

3. The study leads me to believe that Hodgkin's disease is a process to be classified with malignant tumors.

The subject of this paper was suggested to me by Dr. Wm. Ophuls, who noticed a similarity in the histological structure between several cases of Hodgkin's disease and of so-called lymphosarcoma, which he had examined in the laboratory of Cooper Medical College. I am also indebted to him for the use of his material and for most valuable guidance in my work. I am also indebted for the use of their cases, and of clinical notes, to Dr. Emmet Rixford, Dr. J. O. Hirschfelder, Dr. Coffee, and for the autopsy report of one case to Dr. A. E. Taylor.

No attempt has been made to go deeply into the literature of the subject and among the vast number of articles which have appeared upon the subject, only a comparatively few have been mentioned.

SUMMARY OF CASES.

CASE I. (Clinical report in *Occidental Medical Times*, January, 1904.)—Male aged thirty-eight years; native of Italy; varnisher. History of "chills and fever" at periods for three years. From September to December, 1903, three exacerbations of temperature (99° to 105.5°), lasting three to eight days, followed by apyretic (96° to 98°) periods of fourteen to twenty-three days. Blood was normal except for gradual decrease in hemoglobin and a number of red blood corpuscles. Gradual enlargement of all lymph glands, beginning with cervical. Spleen and liver enlarged. Died August, 1904. *Autopsy:* Hodgkin's disease with metastasis in the cervical, axillary, left epitrochlear, right femoral, mediastinal, peribronchial lymph glands, liver and spleen. Glands soft, cut surface white with yellow spots, sections show capsule composed of many fibrous bands including cells, inner bands infiltrated with tumor. Connective-tissue bands through pulp enclosing lymphocytes, epithelioid cells, and a few characteristic giant cells. Nodules in liver and spleen the same. In liver wall of the vein infiltrated with tumor.

CASE II.—Entered Hospital February 13, 1902. Died February 18, 1902. Man, aged thirty-three years; single; upholsterer. First symptoms one month before admission. Loss of appetite, weight, and strength. Gradual enlargement of glands in neck, axilla, and groin. Liver and spleen enlarged. No temperature. Blood, slight anemia. Urine negative. *Autopsy:* Hodgkin's disease, with metastases in liver, pancreas, kidneys, spleen, and enlargement of cervical, intraclavicular, left femoral, left axillary, peribronchial, and retroperitoneal lymph glands. Lymph glands and metastases show the same microscopic and macroscopic appearance. Tissue soft, and shows rapid growth with many new lymph cells. Few giant cells and masses of epithelioid cells. Little connective tissue. Capsule of glands infiltrated and tumor mass pushing through capsule. Wall of vein in liver destroyed by infiltrating tumor cells.

CASE III.—(Clinical report in *Occidental Medical Times*, vol. xvii, p. 443.)—Male, aged forty-three years; single. Eight months before death cervical glands began to enlarge, following severe cold. Followed by successive rapid enlargement of glands all over body. Enlargement of liver and spleen. Rapid loss of weight and strength. Blood showed secondary anemia, otherwise normal. Urine negative. Died October 27, 1903. *Autopsy:* Hodgkin's disease with enlargement of cervical, axillary, inguinal, saphenous, mediastinal, and retroperitoneal lymph glands. Metastases in liver, spleen, and kidney. Lymph glands are soft and on section show recent rapid growth with very little connective tissue. Many lymphocytes and few epithelioid giant cells. Capsule infiltrated and containing masses of tumor cells. Vein in liver shows destruction of wall by advancing tumor.

CASE IV.—A. M. C., man, aged sixty-five years; millwright. In December, 1903, right cervical glands began to enlarge gradually. Reached size of orange and were removed, February, 1904. In one month growth returned and was followed by successive, rapid, enormous enlargement of all external glands. Glands from axillæ removed September 13, 1904. Liver and spleen not enlarged. Died September 28. Glands in the axilla form a mass the size of two fists: glands size of walnut, soft, grayish-white; for most part separate. Some joined by connections of pulp. Cut surface uniform. Pulp bulges from capsule. Section shows similar structure to Case III.

CASE V.—A. W., boy, aged eleven years. In October, 1900, noticed a number of small swellings on right side of neck. These increased in size and number until October, 1901, when ten glands were removed. Four months later recurrence took place accompanied by similar growth on left side and in axillæ; all were again removed. Nine months later recurrence had again taken place and glands were a third time removed from neck, and boy was lost track of. Glands very hard, discrete, largest 2 cm. in diameter, pulp yellowish, crossed by white bands. Capsule evry thick. Section shows thick bands

of dense connective tissue travelling from capsule through pulp with areas of cellular elements between. Histology of gland entirely destroyed. Capsule consists of dense connective-tissue bands with tumor cells between; lower layers infiltrated by cells. Several areas of necrosis.

CASE VI.—C. B., man, aged thirty-four years; native of Ireland; police officer. About January, 1900, small swellings appeared on both sides of neck; a few months later appeared in left axilla. Gradually became the size of an apple and were all removed October, 1900. In three months there was a recurrence, and patient died a year later with all superficial glands and spleen much enlarged. Glands rather hard, show slow growth, with much connective tissue and islands of cellular elements. Many giant cells, marked infiltration of capsule.

CASE VII.—Clinical notes not available, but symptoms were those of Hodgkin's disease. *Autopsy:* Hodgkin's disease with tumor in thymus gland with extension to pericardium and anterior part of left upper lobe. Numerous metastases in lungs; peribronchial, cervical, anterior and posterior mediastinal lymph glands, and in liver and spleen. Tumor, lymph glands, and metastases in organs exhibit same histological picture as in other cases, characterized by considerable connective tissue, with cellular islets between containing lymphocytes, epithelioid cells, and many large characteristic giant cells.

CASE VIII.—Mr. K. In May, 1902, glands on left side of neck began to enlarge; the enlargement progressed until tumor reached size of fist and was adherent to deep structures. Submental glands and right cervical glands became involved later. Died January, 1903, of intercurrent pyemia. *Autopsy:* Hodgkin's disease with tumor involving entire left side of neck, submental, right submaxillary glands, and those about arch of aorta and in mesentery. Tumor grayish white, hard, infiltrating cervical vertebra, sternomastoid muscle, salivary glands, and passing into mediastinum infiltrating wall of jugular vein and pericardium. Tumor and lymph glands show same histological structure, bands of connective tissue including islands of cells, lymphocytes, epithelioid cells, and characteristic giant cells.

CASE IX.—H. S. M., man aged forty years. Nine months before death in February, 1905, enlarged gland appeared on right side of neck, which was removed. Two months later many other tumors developed on same side and grew to enormous size. Glands on left side also enlarged. Gradual loss in weight and strength. At autopsy, tumor on right side of neck, consisting of discrete nodules on outside, tumor size of egg in centre infiltrating muscles and salivary glands, extending through upper aperture of thorax to tumor in anterior mediastinum. No metastases found in organs. Sections of tumor and glands show characteristic histological picture of fibrous type.

THE COAGULABILITY OF THE BLOOD IN YELLOW FEVER.

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Due to the high degree of perfection attained in modern pathological technique, made possible by the improved microscope, by numerous specially devised accurate instruments, and by many newly created chemical stains, medical men of the last two decades have been able to establish the correctness or erroneousness of many etiological and pathological theories accepted in the past. The great majority of these antiquated theories, which were based, for the most part, on macroscopic examination alone, have been found to be grievously wanting, and these have been replaced by scientific facts. However, some of these theories, prominent among which is that of the totally lost or greatly deficient coagulability of the blood in yellow fever, still remain with us, not because they are believed, but because the finishing touches have not as yet been put to the facts that are to cause their final relegation to the domain of the historian.

The majority of the early writers believed that in yellow fever, and especially in the latter stage, the histological elements of the blood were completely destroyed, that, in the greater number of cases, the blood was always carbonized, and that its fluidity was greatly increased. From these conjectures they concluded that the power of coagulation was either completely lost or most tardily established. The following extracts have been selected from the vast amount of literature on this subject, spread out over a period extending from 1793 almost to the present day.

Dr. Rush, a Philadelphia physician, reports eleven different appearances presented by the blood of yellow fever patients in that city during the epidemics of 1793 to 1794. Three of these appearances, being pertinent to this paper, are given herewith: (1) "In the greater number of cases it (the blood) was without any separation into crassamentum (clot) and serum. (2) In some cases there was separation into crassamentum and yellow serum. (3) There was in every case in which the blood was not dissolved, or in which the second appearance, that has been mentioned, did not take place, a beautiful scarlet colored sediment in the bottom of the bowl, forming lines or a large circle. It seemed to be a tendency of the blood to dissolution."

Nassy speaks of "the fluidity of the blood."

Dr. Cathrall, in his treatise on the above epidemic (1794), remarks that "the blood, drawn in the early stage, very seldom separates into serum and crassamentum; when it does, the former is nearly of a natural color, though small in quantity in proportion to the latter."

Dr. Caldwell found in the epidemic in Philadelphia in 1805 that "the blood which flowed in hemorrhage was never capable of firm coagulation, thereby evidencing that its vitality was nearly extinguished."

La Roche[1] accounts for the hemorrhagic tendency in yellow fever thus: "The frequency of hemorrhage in yellow fever, the tendency to effusion, especially during the later stage of the disease, from the several outlets of the body, and through textures, and into parts which, in the natural state and in diseases generally, do not afford passage to blood, must depend upon a general cause, and that cause no one acquainted with yellow fever will fail to detect in the particular alteration of the blood."

Joseph Jones,[2] in his work on yellow fever, published in 1894, makes this statement: "In the marked diminution of fibrin we have an important explanation of the cause of the hemorrhagic tendency in yellow fever."

La Roche, in summarizing, adds: "Similar observations, as the above (those of Drs. Rush, Cathrall, Caldwell, and others), as to the fluidity and defective coagulation and dark color of the blood were made during the fever of 1820 in Philadelphia. But, on this occasion, it was discovered that, although the blood presented these appearances, and, when examined after death, was found in a fluid state, its power of coagulation was often rather tardily established than lost."

Pariset remarks on an epidemic at Barcelona in 1821: "The blood found in the cavities of the heart is black and fluid. If clots exist they are small and diffluent. The blood is apparently deprived of fibrin and no longer red. No longer separating itself into a solid part and yellowish serum. And always remaining black, carbonized, and fluid. The blood is certainly more fluid and black."

Desportes, in commenting upon the yellow fever in San Domingo, states: "The blood obtained by venesection is always very red, florid, and frothy, containing but little serum. If we bleed after the cessation of the fever the blood remains a long time in a liquid state, sometimes three or four hours after the operation, and there is no serum."

Hillary states that "in the last stages the blood is so attenuated and dissolved as to flow from different parts."

Dr. Stevens, a West Indian observer, adds to the above: "The blood found after death has the character of a dissolved fluid, nearly as thin as water, almost as black as ink, and evidently so diseased as to be totally incapable of either stimulating the heart or supporting life."

[1] Yellow Fever considered in its Historical, Pathological, Etiological, and Therapeutical Relations, vol. i. Philadelphia, 1855.

[2] Original Investigations on the Natural History (Symptoms and Pathology) of Yellow Fever, 1854–1894. Chicago, 1894.

Bienparthy, in 1844, gave new life to the dissolution theory by stating that under the microscope "the corpuscles disintegrated, broke down, and dissolved in the fluid plasma."

In order successfully and conclusively to refute the above statements, as to the fluidity, destruction, carbonization, and total loss of the power of coagulation of the blood, it will only be necessary to give place hereunder to the findings of contemporaneous and of later writers.

Dr. Kelly, another West Indian observer, tells us: "I have seen the blood coagulate as perfectly in yellow fever as in ordinary intermittent fever."

Henson made the following experiments in 1820: "I collected portions of blood in cups, in the course of ten to fifteen minutes it was firmly coagulated, and this was found in subsequent observations invariably to occur."

Lawson, in Jamaica, as early as 1802, examined the blood of yellow fever patients, and concluded: "This theory of disorganization has no foundation. The blood is not dissolved; on the contrary, the corpuscles appear entire and well-shaped."

Prof. Joseph Leidy, one of the most eminent scientists of the nineteenth century, stated in 1854: "I have not been able to discover the slightest evidence of the destructive process suffered by the morphological elements of the blood in this disease, as described by Bienparthy."

Sternberg[1] observed in 1879, in Cuba, that "both the red and white corpuscles retained their normal appearance, and I have frequently seen the leukocytes undergoing their characteristic movements, even after twenty-four hours, in blood which had been preserved in culture cells."

Dr. J. Crevaux, a French naval surgeon, studied the blood microscopically in French Guiana in 1877. He states clearly and definitely that he could find "no unusual alteration in the blood corpuscles."

Dr. Carlos Finlay, of Havana, in writing on this subject, in 1882, stated that "not only is the integrity of the blood corpuscles maintained in yellow fever, but their number is apparently increased during the course of the malady." Dr. Finlay believed at this early date that the hemorrhagic tendency was due to a lesion of the vascular endothelium, which he considered the characteristic lesion of yellow fever.

Dr. H. D. Schmidt,[2] pathologist of the Charity Hospital, New Orleans, and one of the authorities of his day on the pathology of yellow fever, concludes from his extensive experience that "it must be admitted that the condition in which its (the blood's)

[1] Yellow Fever, in Reference Handbook of the Medical Sciences, 1894, vol. viii.
[2] The Pathology and Treatment of Yellow Fever, Chicago, 1881.

morphological elements were found actually offers nothing remarkable or otherwise which in any way could be interpreted as peculiar or characteristic of yellow fever."

To confirm the findings of Lawson, Sternberg, Crevaux, Leidy, Finlay, and Schmidt, I might record here that in over twenty thousand examinations of fresh and stain preparations of blood of yellow fever patients made by the various workers at the Emergency Hospital in New Orleans during the epidemic of 1905 not one specimen showed the slightest evidence whatsoever of corpuscular degeneration.

The view of Finlay on the causation of the hemorrhagic tendency in yellow fever is almost proved, while the views of La Roche and Jones on this same subject are completely overthrown by Sternberg's most sensible remark, that "the hemorrhages are due to the disorganized and diffluent condition of the blood a moment's reflection should show, that this explanation is insufficient, and that the blood, however diffluent, cannot escape so long as the vessels are intact." The above statement of Sternberg is itself transformed into a fact by the knowledge which we possess to-day of the fearful damage inflicted by this disease, especially upon the smaller bloodvessels and capillaries of the various organs throughout the body.

I shall now proceed to the remaining question, of "the tardy coagulability of the blood."

La Roche after perusal of all the available literature concluded: "The inability or reluctance to coagulation, though generally exhibited, is not universal, and cases occur in which the blood coagulates with as much readiness and firmness as in other diseases."

From the works of Dr. Jones, we gather: "The following are the chief characteristics of the blood in the stage of depression in yellow fever: Specific gravity of blood and serum not specially altered. Blood coagulates slowly and imperfectly. Clot voluminous and soft. Fibrin quantity deficient and not more than one-tenth the normal amount. Reaction of blood alkaline," etc.

In order to refute the above, I shall first state briefly our macroscopic findings, and then record the results attained by actual experiments with an accurate instrument.

1. I have repeatedly seen blood drawn from the median vein of a yellow fever patient coagulate before it could be forced out of the aspirator into a receptacle held ready to receive it—the blood not remaining in the aspirator more than from three to five minutes.

2. Blood drawn from the median vein of a yellow fever patient and placed in a large test-tube alongside of another similar tube containing normal blood, drawn in the same manner, did not differ in the slightest degree from the latter, and one not knowing which was which could not so state.

3. In performing certain filtration experiments, it was found impossible to filter unaltered blood, and to overcome this readiness

to coagulate, it was necessary to add a solution of potassium oxalate to prevent rapid coagulation.

In making the actual experiments, Wright's coagulometer was used. Space will not be consumed here in describing the technique employed, as this can be found in almost any standard text-book on clinical diagnosis. However, a record must be made herewith that the heating chamber of this instrument was discarded, since the temperature prevailing in the city at the season of the year during which these tests were made was fairly constant and not sufficiently below the standard as to affect materially the results. Eight of the most typical cases of yellow fever were selected for these tests, with the following results:

In speaking of symptoms existing prior to tests, I shall only record those which are considered characteristic of the disease, and make no mention of pain, temperature, etc.

CASE I.—M. P., white, male, aged thirty-five years; nativity, France; occupation, barkeeper. One year in New Orleans. Symptoms prior to test: Injected sclera, general jaundice, spongy and bleeding gums, profuse epistaxis. Vomited at intervals large quantities of unaltered blood. Maximum amount of albumin, 13 per cent. (Purdy's method.) Bile-stained casts.

Test No. 1 on twelfth day of illness: Coagulation was negative in three minutes and thirty seconds; positive in four minutes.

Test No. 2 on fourteenth day of illness: Coagulation was negative in three minutes and thirty seconds; positive in four minutes.

CASE II.—F. W., white, male, aged forty-three years; nativity, Texas; occupation, blacksmith. Symptoms prior to test: Jaundiced sclera, gums spongy, afterward bleeding; maximum amount of albumin, 6.5 per cent.; bile-stained casts later in disease.

Test No. 1 on second day of illness: Coagulation was negative in two minutes and forty-five seconds; positive in three minutes (four tubes).

Test No 2 on sixth day of illness: Coagulation was negative in three minutes and thirty seconds; positive in four minutes (six tubes).

CASE III.—J. S., white, male, aged twenty-three years; native of New Orleans. Symptoms prior to test: General jaundice, spongy and bleeding gums. Maximum amount of albumin—trace.

Test No. 1 on fifth day of illness: Coagulation was negative in three minutes and ten seconds; positive in four minutes (six tubes).

CASE IV.—E. J. M., white, male, aged twenty years; native of New Orleans. Occupation, boilermaker. Symptoms prior to test: Injected sclera, general jaundice, profuse epistaxis, bleeding gums, black vomit. Maximum amount of albumin, 14 per cent.; bile-stained casts.

Test No. 1 on seventh day of illness, one-half hour after black

vomit. Coagulation was negative in three minutes and ten seconds; positive in four minutes and ten seconds.

Test No. 2 on ninth day of illness: Coagulation was negative in three minutes and eleven seconds; positive in four minutes.

CASE V.—J. B., white, male, aged twenty-six years; native of New Orleans. Symptoms prior to test: General jaundice, bleeding gums, black vomit (once), slight renal suppression. Maximum amount of albumin, 5 per cent.; large number of casts, *not* bile-stained.

Test No. 1 on sixth day of illness: Coagulation was negative in three minutes and thirty seconds; positive in four minutes and twenty seconds.

CASE VI.—A. L. B., white, male, aged twenty-eight years; native of Louisiana; occupation, clerk. Symptoms prior to test: Bleeding gums, slight jaundice, injected sclera. Maximum amount of albumin, 7 per cent.; bile-stained casts.

Test No. 1 on third day of illness: Coagulation was negative in three minutes; positive in three minutes and forty-five seconds.

CASE VII.—F. A., white, male, aged twenty-one years; nativity, France; occupation, tramp. Walked into New Orleans twelve days before beginning of attack. Symptoms prior to test: Spongy gums, eyes injected. Maximum amount of albumin, 2.5 per cent. Vomited several times; unaltered blood. Large number of bile-stained casts.

Test No. 1 on eleventh day of illness: Coagulation was negative in three minutes and ten seconds; positive in four minutes.

Test No. 2 on twelfth day of illness: Coagulation was negative in three minutes and ten seconds; positive in three minutes and forty seconds.

CASE VIII.—C. L., white, male, aged thirty years; nativity, Germany. Symptoms prior to test: Marked jaundice, gums spongy and bleeding, vomited once on eighth day small amount of black vomit. Maximum amount of albumin, 4.5 per cent.; bile-stained casts.

Test No. 1 on eleventh day of illness: Coagulation was negative in three minutes; positive in three minutes and thirty seconds (five tubes).

A brief summary of the foregoing shows: Minimum time of positive coagulation, two minutes and forty-five seconds; maximum, four minutes and twenty seconds; average, three minutes and fifty-two seconds.

From these findings I can state almost without hesitancy that the coagulability of the blood in yellow fever is normal.

I acknowledge the kind direction and valuable assistance rendered me, while performing this work, by Drs. J. T. Halsey and P. E. Archinard, and many others.

UNILATERAL PARALYTIC CHOREA, WITH A REPORT OF A CASE.[1]

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Of all nervous disorders chorea is probably the most easily diagnosed, for the reason that the symptoms are so striking and characteristic. This statement, however, applies only to the ordinary form of the disease, with the irregular muscular twitchings, the constant unrest and nervousness that are always found in a well-developed case. In the majority of instances the symptoms are so pronounced as to enable the teacher or the mother of the little patient to diagnose St. Vitus' dance even before the physician makes the diagnosis of chorea.

It is quite different, however, in the cases of chorea with ill-defined symptoms when the disease is just developing, and in the cases in which paralytic symptoms predominate over the irritative ones, viz., the jactitations and twitchings. The diagnosis is most difficult and almost impossible when the paralytic symptoms confine themselves to one side of the body and are unaccompanied by the spontaneous and inco-ordinate movements so commonly seen in chorea.

Fortunately these cases are rare, and the diagnosis can be made either by exclusion or by attention to some points ordinarily found in most cases of chorea but disregarded. It is with a view of emphasizing these that I report a rather rare case of unilateral paralytic chorea with a peculiar etiology.

As far back as 1850 the great Todd[2] recognized paralytic chorea, which he describes as follows: "In a large proportion of cases of chorea the choreic movements occur more on one side than on the other, and sometimes they will be altogether confined to one side—the child being hemiplegically affected. In such a case, as happens when the choreic movements have wholly or partially subsided, the limbs that were previously twitching now become paralyzed. The case now resembles in every detail that of ordinary hemiplegia from cerebral causes. But the following points of difference can usually be observed:

"1. The face is often not affected, or slightly so.

"2. There will not be any paralysis of the tongue, but more or less of the peculiar mode of protrusion which characterizes chorea.

"3. The paralyzed limbs will exhibit, even in a very slight degree, the choreic movements."

[1] Read at a meeting of the Chicago Neurological Society, March, 1906.

[2] Clinical Lectures on Paralysis, 1850, p. 313.

Gowers[1] reports five cases of paralytic chorea in which one arm was usually more affected than the opposite one, and in which seven to fourteen years was the age of predilection. He says: "Whenever a child of this age suffers from gradual loss of power in the arm and presents no weakness in the face, tongue, or leg, the disease as far as I have seen is always chorea. If the nature of the case be suspected, confirmatory evidence—slight occasional spasm—if looked for, will commonly be at some time detected, either in the weak arm or in the other. The course of these cases is tedious, and as the choreic movements may increase as the weakness passes off, the patients often seem, to the friends, to be getting worse when really getting better. I have never seen this form pass on into severe general chorea."

Osler[2] says: "Muscular weakness is a prominent symptom of chorea and may result in a condition of paresis or even paralysis. It may be hemiplegic, paraplegic, or—more frequently—monoplegic. Very rarely does the loss of power reach the grade of complete paralysis, but the child may be unable to lift the arm to the head, a shoulder may droop, the grasp may be feeble, or there is wrist-drop. It is important to remember that there may be extreme paresis, with slight inco-ordination and few spontaneous movements. Weakness of the arm is shown by enfeebled grip, of the leg by a dragging or by a decided limp. The paralytic symptoms may precede or come on in the course of the attack, more rarely follow it."

The cited descriptions of paralytic chorea are certainly clear and should help one to make a diagnosis in every case. However, if the disease appears after a trauma and as a pure hemiparesis, without choreic twitchings, even the lucid description of the masters will leave us in doubt unless we also consider the points brought out by later investigators. In my opinion, O. Förster[3] has given the best analysis of the motor disturbances in chorea. He divides them into (1) spontaneous choreic movements, and (2) inco-ordination of voluntary movements. But I believe we can conveniently subdivide the last heading into (*a*) inco-ordination proper, and (*b*) muscular weakness.

1. The spontaneous choreic twitchings are irregular jerks; that is, the elements of rhythm and regularity, such as are found in tremors, are always absent in true chorea. Further, the twitchings mostly occur in single muscles, or in muscle groups that do not act synergetically, as opposed to other forms of muscular movements that preferably affect entire muscle groups which constantly act in unison. The spontaneous movements of chorea never resemble a voluntary movement and seem entirely devoid of purpose.

[1] British Medical Journal, 1886, p. 636.

[2] Medical News, 1887, p. 466.

[3] Das Wesen der Choreatischen Bewegungsstörung. Volkmann's Sammlung klin. Vorträge, October, 1904.

This distinguishes the disease from tic, in which condition movements, though involuntarily executed, have a superficial resemblance to a purposive act. The choreic twitchings may be likened to the clonic contractions in epilepsy as regards the suddenness of their occurrence. They differ, however, in the fact that there may be only one or two jerks followed by complete relaxation in chorea, while in epilepsy the clonic spasms are an accession of a series of muscular contractions upon muscles already in tonic contraction, which contractions continue for some little time and only gradually merge into relaxation.

2. (*a*) *Disturbances of Co-ordination.* There is a lack of promptness and instability in muscle innervation. The impulse is either delayed or side-tracked into byways instead of reaching the intended muscle paths. Or, if the intended movement is executed there are so many accessory irrelevant movements that an inco-ordinated act is the result instead of the orderly co-ordinated intentional movement of health.

(*b*) *Muscular weakness* results from either a delayed nerve impulse or from dissipation of well-directed energy into channels having an inhibitive effect upon the intended movement.

To summarize this analysis, we have, on the one hand, the involuntary so-called choreic spontaneous movements which depend upon an irritation of single muscles and are dissimilar to voluntary movements. On the other hand, we have disturbances of co-ordination; that is, a disturbance either in the execution of voluntary movements or in the maintenance of static equilibrium. The inco-ordination manifests itself in its complete form as follows: The principal muscles concerned in voluntary movements are promptly innervated, but the impulse is miscarried to a varying degree. In severe cases it is so ineffectual as to cause distinct paresis affecting equally all the muscles of an extremity. In the simple movements of the fingers, tongue, and lips the innervation may be delayed and intermittent.

The impulse for the so-called agonists, the muscles directly concerned in a movement, as opposed to the antagonists, which merely assist the agonists, may radiate into other channels or along the commissural tracts to the opposite side; then we have purposeless associated movements. In like manner the antagonists that support or maintain the agonists are not properly innervated in severe cases of chorea and we have intermittent muscular contractions. In a general way we may state that those impulses that are directly concerned in the volition of station or locomotion are functionating properly; in fact, there is even an excess of function leading to associated movements, while those involuntary motor impulses concerned in the maintenance of equilibration which emanate from the cerebellum and its paths are markedly deficient in chorea. This

has led Otfrid Förster to formulate a theory that chorea is primarily a disease of the cerebellum or of the superior cerebellar peduncles.

We may assume with him that the toxic agents causing chorea act principally upon the cerebellar tissue and produce both irritant and paralytic effects. The irritant impulses upon the muscles are probably conducted from the cerebellum along the superior cerebellar peduncle, through the red nucleus, to be transmitted either directly into the centrifugal paths, or else to be carried up still higher through intermediate stations into the cerebrum, whence the motor impulse is transmitted in the same way as the twitchings of symptomatic chorea and epilepsy.

Whether this theory proves correct or will shortly be substituted by some other theory, it certainly explains most of the phenomena found in chorea, and particularly does it throw some light upon paralytic chorea.

A study of the various movements in chorea led Weir Mitchell and J. H. Wallace Rhein[1] to distinguish four different types:

1. Movements are continuous during the period of muscular inaction. Hands lying in patient's lap are in constant motion. But when patients perform a delicate muscular effort there is an entire disappearance of movements lasting throughout the muscular effort.

2. In this type there are clonic contractions during rest. Volition so aggravates them that a voluntary movement cannot be accomplished. There is a lessened inhibition during willed muscular effort. This is the usual type.

3. The rarest variety becomes evident when patients attempt to perform a muscular act. The hands at rest move, if at all, only slightly and at rare intervals, but on attempting to use them the twitchings become sufficiently active to prevent or greatly to interfere with the performance of the act.

4. There is a small number of cases in which the movements, continuous during rest, are but slightly altered by the tests employed.

My case showed the third type of movements during the latter part of its course.

Case.—J. B., aged seven years, a school-boy of Irish-American parentage, was first seen by me January 12, 1906.

Family history is negative: father, mother, brother, sister, uncles, and aunts are free from nervous and mental disorders.

Patient was born in normal labor, walked and talked in proper time, never had convulsions or other nervous manifestations, and mental development showed no anomalies. Of past illnesses, he had diphtheria, followed by mumps, about a year ago, and an intestinal affection, resembling typhoid fever, about nine months ago.

[1] Philadelphia Medical Journal, 1898, p. 153.

He never had rheumatism or growing pains, nor was there an acute sore throat prior to the development of present complaint.

Present Trouble. About three and a half months ago while on his way from school a companion kicked him in the region of the right ankle. A soft swelling soon appeared over the outer malleolus and the boy began to limp. When he reached home his mother massaged the affected parts and the swelling promptly subsided, but the limping continued. Within the next few days a weakness developed in the entire right lower extremity and a few days later the right upper extremity became similarly involved. All movements with the right arm and hand became clumsy and subsequently impossible.

Four weeks after the beginning of these symptoms he came under my observation.

Examination. The patient, a healthy appearing boy of medium height and weight, is free from signs of visceral disease. His gait, however, is defective, in that he drags his right leg, resembling somewhat the polyneuritic step. The right arm hangs passively by the side and dangles somewhat when the patient walks, so that he gives one the impression of being the subject of a right-sided hemiplegia. The face shows no weakness or paralysis. The pupils are equal and respond promptly to light and accommodation.

Upon testing muscular strength a uniform diminution was found in the flexors and extensors of the upper and the lower extremity on the right side, more of a paresis than a paralysis. The grip in the left hand was good, but that of the right hand was almost nothing, despite the little patient's strenuous efforts to force up the lever of the dynamometer. Neither signs of injury nor atrophies were discoverable anywhere upon repeated examination.

The superficial and deep reflexes appeared normal on both sides, except the right knee-jerk and Achilles tap, which were slightly reduced. In eliciting the patellar reflex I found the upstroke very prompt, while the downward movement was considerably delayed bilaterally, particularly on the paretic side. Upon tapping the tendon the limb flew up promptly, then dallied a while, and came down in a zig-zag manner, continuing this rhythmic movement even after it had regained its former position. There was no spasticity on the affected side and no Babinski, Oppenheim, or Gordon signs. The tongue was protruded in a straight line and showed a few irregular twitchings. No sensory disturbances were found.

About three weeks after my first examination a slight improvement was noticed, but when the patient attempted to use his right hand and arm a peculiar jerkiness and tremor appeared, resembling partly the intention tremor of multiple sclerosis, and having some of the characteristics of tabetic inco-ordination. When directed to touch his nose with the tip of the right index finger there were several purposeless excursions in the arm, forearm, and finger, becom-

ing more exaggerated when he had almost accomplished his design. In an attempt to grip my hand with his right, at first some power was developed, which dwindled down to nothing; then alternating contraction and relaxation of muscles occurred, which could not only be felt but also seen as the tendons intermittently sprang into view. In raising his right lower extremity from the ground inco-ordinate movements developed at once and continued as long as the effort was maintained. In walking he appeared to throw out his right foot instead of using dorsal flexion. The muscular twitchings, previously barely noticeable, had become more numerous. At a subsequent visit the symptom of associated movements could also be demonstrated. When he attempted to make a fist with his left (the unaffected) hand the right participated; when he raised his left leg the right also ascended. But when the movement was initiated by any of the extremities of the right (affected) side no similar participation was indulged in by the left-sided members.

At present his paralytic symptoms are on the decline, so that he walks better and is regaining the use of his right hand. The choreic twitchings, however, have become more marked. This, according to most observers, is a favorable sign.

To summarize: We have a hemiparesis which developed after slight trauma in a previously healthy boy. The weakness began with limping, and within ten days slowly spread over the entire right half of the body with the exception of the face. The reflexes were slightly reduced on the affected side; there were no atrophies, spasticity, or sensory disturbances.

In the differential diagnosis we must consider cerebral hemiplegia, neuritis, and poliomyelitis. Against the diagnosis of cerebral hemiplegia speak the absence of spastic symptoms and the etiology. Anterior poliomyelitis can be ruled out because of the absence of atrophies. The same course can be pursued with neuritis, in which sensory disturbances are a marked feature, while they were never present in this case. We must now consider hysteria and chorea. The patient shows no ancestral or personal tendency to the great neurosis, being of sturdy Irish-American stock, and one out of six young children who are not reared in luxury and receive only the little attention that a workingman's wife can bestow upon them. The boy never showed signs of nervousness and a careful search for hysterical stigmata was negative. Even by exclusion we are forced to make the diagnosis of paralytic chorea. But how can we account for the peculiar onset of the disease with a trauma? As in all forms of trauma, we have the physical and psychical elements to reckon with. The physical element in this case was almost *nil*, a slight transient swelling over the ankle; the psychical element, the unexpected attack—the fright—precipitated the disease, as it often does in other cases of chorea. The physical trauma may have determined the beginning of the

condition at the site of its occurrence, but was of secondary importance otherwise.

As regards *treatment*, rest, tonics, and a modified form of Fränkel exercises are the only measures now employed. In the beginning I ordered arsenic in the form of Fowler's solution, which had to be discontinued because it affected the patient unfavorably. Antipyrine in five-grain doses was then substituted, with doubtful results; he then took hypophosphites, with marked benefit. By July, 1906, the patient showed great improvement; he was able to use his right hand, and the gait was very nearly normal. There was only an insignificant amount of inco-ordination in the right upper and lower extremities, and no twitchings could be observed anywhere. The course in my case is rather prolonged (since December, 1905), but, like other prolonged cases, tends toward complete recovery.

In conclusion, I wish to emphasize that associated movements should be looked for in every case of supposed chorea, that some degree of inco-ordination is almost a constant symptom in this disease, and that various degrees of muscle weakness will be found. Muscular twitchings, be they ever so few, constitute a valuable diagnostic aid. A helpful symptom in doubtful cases is the knee-jerk retarded in its descent, first studied and described by Gordon. The slow development of a paralysis in an otherwise healthy child should make us think strongly of paralytic chorea.

POSTURAL TREATMENT OF OTITIS MEDIA AND MASTOIDITIS.

By A. E. Schmitt, M.D.,

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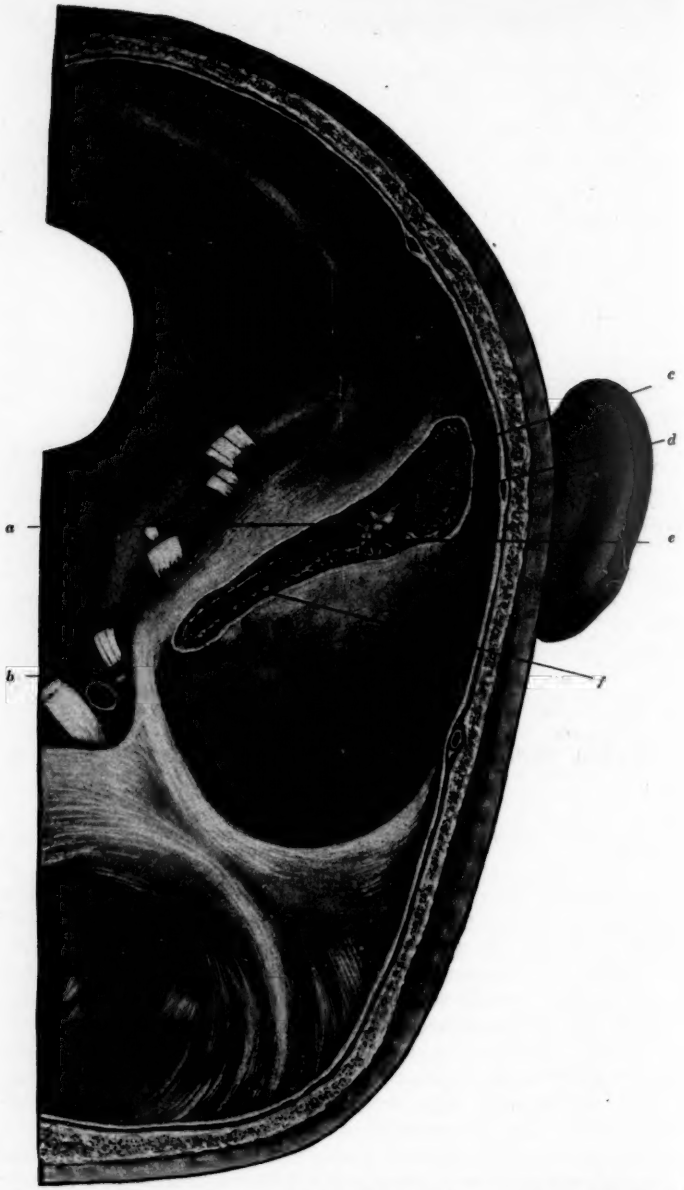
During the past few years operative procedure has become so common in otology that with many surgeons the slightest sign of involvement of the mastoid antrum complicating an otitis media is considered an indication for operative interference. A better understanding of the anatomical relations of the parts, together with the application of one of the fundamental principles of successful surgery, that is, drainage, would lead to a more conservative line of treatment in a direction that seems to have been entirely overlooked, namely, the posture of a patient with otitis media or mastoiditis. The relationship of the middle ear and antrum is such that, if an inflammatory exudate is present in the middle ear and the patient is lying on his back, the secretions will flow by gravity from the middle ear through the aditus ad antrum to the mastoid antrum itself. The presence of a secretion will, of course, irritate the membrane lining the antrum and produce mastoiditis,



Base of the skull seen from above with the dura mater intact, except over a portion of the temporal bone where it and the bone have been cut away to expose the Eustachian tube, the tympanic activity, the attic, aditus ad antrum, and the mastoid antrum. *a*. Long process of malleus. *b*. Carotid artery. *c*. Mastoid antrum. *d*. Short process of the incus. *e*. Tympanic membrane, with perforation. *f*. Eustachian tube.

even though the inflammation has not extended from the middle ear. Let us recall for a moment the exact relations of the parts in the tympanic cavity. (See accompanying figures.)

Anteriorly is the opening of the Eustachian tube. It is through this tube that an inflammation originating in the nose or pharynx extends and causes an involvement of the middle ear. The mucous membrane lining the tube swells and clogs this channel temporarily. The natural passage through which the middle ear is aërated and drained is closed, and the result is a retention of the inflammatory secretions within the middle ear, which aggravates the already inflamed membrane. If not relieved there is in time a bulging of the tympanic membrane accompanied by great pain, and if a paracentesis is not performed an extension to attic, aditus, and antrum occurs. If the drainage through the perforation is sufficient, the process of repair sets in and frequently the inflammation of the entire tract subsides, even though it has extended to the attic, aditus, and antrum. If pus is present, there is an erosion of the membrane which, owing to interference of the circulation from pressure, has become less resistant, and a perforation results—which gives relief from the symptoms of pressure. As far as the middle ear is concerned, the tendency now is to repair. But what happens to the antrum if it has become filled with the exudate? As these cases are now ordinarily treated, the patient is permitted either to sit erect or lie on the back, or occasionally on his side. In the recumbent position the irritating discharge remains in the antrum and causes an inflammation which, in turn, adds to the exudate, of which only the overflow leaves the antrum through the aditus.

The inflamed membrane goes on to ulceration and eventually to necrosis of the bony lining, with further involvement of efferent veins and lymph channels, infecting all the important surrounding structures and thus establishing the various complications of middle ear disease. If the mastoid process is cancellous, the extension of the inflammation to other pneumatic cells throughout the process may be very rapid. Necrosis extending backward from the antrum soon reaches the bony lining of the lateral sinus, or the inflammation may extend through the connecting veins of the sinus, producing a thrombosis of the lateral sinus, without antecedent necrosis.

Referring again to the anatomical relations, we have externally the drum membrane; internally, the bony inner wall, with exception of the membrane of the round window; above, the ossicles and the bony roof; posteriorly, a bony wall below and an opening above, that is, the aditus or passage which leads to the antrum. The position of this passage high up on the posterior wall undoubtedly in many cases prevents the inflammatory process from reaching the antrum. On the other hand, when the antrum is once involved and the patient is allowed to rest on his back, the position of the aditus in its relation to the antrum is such that the acrid secretions

are prevented from leaving the latter, and thus is produced all the trouble we are familiar with. What then can be done to relieve this apparent faulty provision of nature when inflammation ensues? This apparently faulty provision of nature is really an excellent one and finds its parallel in the accessory sinuses of the nose.

The ostea of the maxillary, frontal, and sphenoidal sinuses also are at the highest point possible and well protected, so that no extraneous matter from the inhaled air can enter and infect them. When, however, inflammation occurs, the ostea are most unfavorably situated for draining the cavities, and if relief is not given by posture they must be opened at the most dependent point. This question of drainage is the all-important factor in the successful management of these cases, and the anatomical relations point out the proper course to pursue. If the middle ear or antrum is to be drained, the patient should be placed in such a position that it will drain. And what is this position? *It is one in which the antrum is placed at a higher elevation than the middle ear, and the middle ear higher than the Eustachian tube, its natural outlet or drain.* With a patient lying face downward—the forehead and one cheek supported by pillows, the nose and mouth and one eye free—perfect drainage can be procured. When the patient is made to realize the importance and advantages of this position, he will not only assume it from necessity, but even adopt it by preference as a comfortable posture during sleep. Now, what happens in this posture?

If an otitis media alone exists, the secretion will drain through the Eustachian tube, provided it is not clogged, or through a perforation of the drum membrane, if such exists. This would prevent antrum involvement, which might have occurred if the secretion had been allowed to flow into it and be retained there. If the antrum is already inflamed, the secretion, flowing off through the aditus into the middle ear and out as before, will enable the mucous membrane lining it to proceed to repair, and erosion of it and necrosis of the underlying bone will not occur. Even though the external symptoms of pain on pressure and œdema over the mastoid process have occurred, generally these will gradually subside and disappear, and an operation with a tedious and painful after-treatment will become unnecessary.

The management of otitis media or mastoiditis is, therefore, as follows:

A purgative is given to aid in relieving the local congestion in the affected area. The rhinitis or pharyngitis, whichever may have been the causative factor in the involvement of the Eustachian tube, receives the utmost attention, the greatest efforts being directed to the Eustachian opening and nasopharynx. Following this, the tube itself is treated in order to reduce the swelling of its lining membrane, to render it patent, and thus effectually to drain the middle ear. The patient is directed to produce suction in the Eustachian

tube by swallowing water, with the nose and mouth closed, several times daily. If these measures do not suffice to relieve the condition in the middle ear without operative procedure, paracentesis of the drum membrane should be performed. Instead of the ordinary puncture of the membrane, I incise it from its midpoint (tip of long process of malleus) downward, and make a second incision posteriorly, beginning half-way up and curving downward and forward until the second and first incisions meet, thus forming a flap. This gives a larger opening than a single incision, and, besides permitting free draining, is large enough for introducing a small cannula for irrigating the middle ear. In douching the tympanic cavity in mastoiditis the stream should be directed upward and backward toward the aditus. The patient is advised to lie face downward most of the time, and occasionally upon the affected side. The Eustachian tube should be treated through the nose by means of a catheter, through which a mild antiseptic and detergent solution may be introduced; this flows out through the perforation in the drum. On the other hand, if in irrigating the tympanic cavity the Eustachian tube is patent, the solution will flow out of the nose or into the throat. For irrigating through a perforation, a thin cannula, bent at the end and attached to a rubber bulb placed at an angle permitting exact introduction by sight, should be used. The irrigations are performed by the attending physician twice daily or at longer intervals according to the stage of the process. This frees the middle ear, the aditus, and the Eustachian tube, and as soon as the secretions have diminished and drainage is sufficient through the Eustachian tube, the flap made in the drum may be allowed to fall back into place and to close the perforation, forming a perfect membrane instead of the large perforations that are liable to result from other incisions. The patient himself douches the outer ear at more frequent intervals, and retains the prone posture for the greater part of the time between treatment. Before douching, the patient is instructed to introduce a detergent and cleansing solution full strength into the outer ear to be retained for from five to ten minutes while lying on the other side.

Adrenalin, alcohol, powders, or other medicaments may be used as indicated, to hasten the process of repair. I am also in the habit of having patients take one-half gallon of some alkaline water during the twenty-four hours, as I think it thins the discharge, causes it to flow off more rapidly, and prevents accumulation and formation of crusts.

In several instances of severe otitis media and mastoiditis, in which, in the opinion of eminent otologists, it was deemed necessary to operate immediately, I have been able by this more conservative treatment to cure the condition present; and I feel that by this simple method, I have prevented many cases of otitis media from developing into operable mastoiditis. Of the measures used the postural treatment was probably the most essential.

PARALYSIS OF THE FACIAL NERVE DUE TO THE EUSTACHIAN ELECTRO-BOUGIE: REPORT OF A CASE.

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A review of the literature of electrolytic dilatation of the Eustachian tube, appearing subsequent to Duel's publication of a new method in 1897[1] and his later reports of cases[2] and results,[3] has failed to reveal an accident similar to the subject of this paper. Nor has any mention been made of the possibility of such a complication in any of the various reports, special text-books on the ear, or in the published transactions of otological sections or societies which have been accessible. The accidents which have been experienced thus far in the procedure have been promptly ventilated, however, in the discussions of otological meetings, and by several of the few authors who have taken issue upon the method. Furthermore, in 1903 Dr. Duel asked by letter for reports upon the results of the method, if used, from some two hundred and fifty otologists, and published the information obtained.[4] He also recently informed me that he had not heard of such a case.

Symptoms referable to irritation of the chorda tympani have been met with in a few instances. I have had two such cases, but as the lesion is distinct from the injury of the main trunk of the facial to be described, it will be reserved for a future report dealing with an experience of over two years with the method.

E. S. F., aged forty years, married, a broker, came under treatment on February 17, 1905. At that time I had employed the method, as elaborated by Dr. Duel, in some fifty selected cases in the aural clinic of Dr. Dench at the New York Eye and Ear Infirmary, and in private practice, with very favorable results, and without accidents other than the temporary irritation of the chorda tympani mentioned. The patient's family history was distinctly neurotic, but otherwise good. His personal history was negative for tuberculosis, syphilis, gout, influenza, autotoxic or infectious arthritis. He was supposed to have had smallpox when ten days old. After an attack of scarlatina in childhood, the date of which is not recalled, he distinctly remembers having the right ear syringed for some days. He had measles at sixteen;

[1] On a New Method of Dilating Strictures of the Eustachian Tube by Means of the Galvanic Current, N. Y. Med. Jour., January 16, 1897.

[2] The Rapid Dilatation of Strictures of the Eustachian Tube by Electrolysis, New York Eye and Ear Infirmary Reports, 1897, vol. vii.

[3] The Value of Electrolytic Dilatation of the Eustachian Tube in Chronic Tubal Catarrh and Chronic Catarrhal Otitis Media, Amer. Jour. Med. Sci., 1900, p. 426.

[4] The Possibilities and Limitations of the Electro-bougie in the Treatment of Chronic Catarrhal Otitis, The Laryngoscope, 1903, p. 522.

otherwise no serious illness, and never any other acute inflammation of the ears. Impairment of hearing, accompanied by tinnitus in both ears, was first recognized twenty-one years ago. Inflation produced no improvement. Ten years ago ossiculectomy was performed on the right ear, relieving the tinnitus and improving the hearing temporarily. Vibratory treatment and inflation were tried four years ago without benefit. Two years ago the hearing and general health became so impaired that the patient was obliged to give up his work, and went abroad to consult foreign otologists. Vibratory treatment was advised and given by a distinguished Münich otologist, and the tubes bougied by the ordinary method, but without appreciable effect upon the hearing or tinnitus. One year ago the tubes were bougied in the usual manner in this country and were declared to be open. Since then the patient has not been under treatment.

Examination. The patient is of a highly nervous temperament; well nourished but pale. Hemoglobin (Fleischl), 80 per cent. Otherwise the general condition shows nothing significant. Mucous membranes of nose and throat congested and hypertrophic. Eustachian eminences spongy and congested. *Right Ear:* Drum thickened by irregular deposits; the tube is patent. Tone-limits, 2048–128. Bone-conduction normal. Watch not heard. Whisper (forced): sibilants at one foot, aspirates at four inches. *Left Ear:* Drum retracted and dull. Light-reflex contracted. Process of malleus prominent. Weber increased, Rinne negative, Swabach lengthened. Tone-limits, 2048–128. Watch not heard. Whisper (forced): sibilants at eighteen inches, aspirates at one foot. Tube admits very little air on inflation.

A medium-sized gold electro-bougie (Duel-Meyrowitz) was, therefore, passed into the left tube under the usual precautions, and 1 milliampère of current admitted from a thirty-cell dry battery. The bougie advanced readily through the membranous portion, but met with some resistance in the bony canal. The latter resistance was fairly uniform, yielding gradually, until the bougie had reached a point one and one-half inches from the pharyngeal opening. It then impinged against what felt like a firm, fibrous constriction. Steady but gentle pressure with 1.5 milliampères of current failed to overcome the obstruction at the end of five minutes, so the attempt was discontinued and the bougie slowly withdrawn. The discomfort of the operation and the crackling sounds experienced in the bony canal were slight. The hyperemic reaction of the drum was not unusual. The hearing remained unchanged. The patient was advised in regard to nasal spray, hygiene of the nose, throat, and teeth, hemic tonic, and general hygiene, and recommended to return on the third day for inflation. He was unable, however, to do so until one week later.

On February 25, the smallest bougie was passed easily, with

1 milliampère of current, to the constriction, one and one-half inches from the mouth of the tube. As the stricture did not differ essentially in situation and feel from many others met with, and furthermore corresponded with the conclusions reached by Bryant[5] in regard to fibrous constrictions, no unusal apprehension was felt in attempting to overcome it under the rigid precautions necessary in view of the proximity of the chorda tympani, the tympanic membrane, and the facial nerve. Firm, gentle pressure was maintained and at the end of a minute the current was increased to 1.5 milliampères. The crackling and the discomfort from the pressure were slight. In about another minute the bougie suddenly advanced about an eighth of an inch and met a firmer resistance, accompanied by sharp pain. The bougie was at once withdrawn one quarter of an inch, when on looking at the patient's face the entire left side was seen to be relaxed. Treatment was immediately discontinued. The left eye could be opened slightly; control of the other muscles on that side of the face was lost. Pain subsequent to the accident was slight and of short duration. The usual amount of hyperemia of the drum was present. On testing the hearing: sibilants were audible at three feet, aspirates at two feet, a gain of eighteen and twelve inches respectively. In about fifteen minutes the left eye could be opened wider and the eyebrow raised slightly. As the prognosis for the paralysis and the hearing was favorable the patient was reassured, especially as he was aware of an immediate diminution in the feeling of stuffiness in the ear and of an increase in the hearing.

February 27th. Paralysis still marked. Hearing unchanged. Tubes were inflated, and, as on all previous and subsequent visits, the mouths of the tubes were swabbed with adrenalin before, and with silver nitrate after the inflation.

March 4th. Paralysis less marked. Tube more open. Hears watch for the first time at one-half inch; sibilants at three and one-half feet, aspirates at two feet.

18*th.* Marked increase in the tone of all the left facial muscles, except the levator palpebræ superioris. Galvanic current applied over left side of face and forehead. Tubes inflated.

25*th.* Slight droop of the upper eyelid and of the left corner of the mouth, otherwise the flattening of the face has disappeared. Galvanism to face. Tubes inflated.

April 1st. Ptosis very slight. Tube is open. Hears sibilants at six feet, aspirates at two feet. Galvanism to face.

8*th.* No evidence of paralysis except sluggish contraction of levator palpebræ superioris in looking up. Tube inflated. Galvanism to face.

15*th.* Before inflation hears sibilants at six feet, aspirates at two feet; after inflation, sibilants at twelve feet, aspirates at four feet. Galvanism to face.

22*d*. Has had sore throat for several days. Mucus in left tube on inflation. Hears: before inflation, sibilants at eight feet, aspirates at three feet; after inflation, sibilants at twenty-five feet, aspirates at ten feet. Galvanism to face (last treatment).

From April 25 to July 28 the patient was obliged to be away on business and was seen only three times. During this interval he had several slight colds, the ear had felt stuffy, and the hearing had diminished. On the three occasions when seen inflation was not free, but as the case could not be followed closely bougieing was contraindicated.

On July 28 sibilants were heard at six feet, aspirates at three feet. Inflation was not free. A medium-sized bougie was accordingly passed with 1 milliampère of current, and advanced one and three-eighths inches from the mouth of the tube. The membranous portion was felt to be congested, but yielded gradually to slow and gentle ironing. The bony canal presented markedly less resistance than when first bougied on February 17. There was but little discomfort, although the patient became very apprehensive as the bougie entered the bony canal and the crackling began. The reaction was slight. The sense of fulness in the ear was at once relieved, but the hearing remained unchanged. In the following months the patient was again called away and was not seen until October 16, when the tube was found to be more open. Sibilants were heard at ten feet, aspirates at four feet. Another interval occurred on November 3, when the left drum was found to be reddened along the long process of the malleus and Schrapnell's membrane. When seen again on December 23, the redness had disappeared, but the tube was closed.

December 27th. Hearing before inflation: sibilants at eighteen feet, aspirates at four feet. After applying adrenalin to the mouth of the tube inflation was free. Sibilants were then audible at twenty-five feet, aspirates at eight feet.

30*th*. Before inflation hears sibilants at twenty-five feet, aspirates at ten feet; after inflation, sibilants at thirty feet, aspirates at twenty-three feet. The watch is audible at one inch. Tone-limits, 2048–26.

From December 30 to January 19, the date of last visit, the patient has been seen at regular intervals. Aspirates were heard at twenty-five feet, and the tube has remained open.

During the eleven months which have elapsed since the injury, the hearing has, therefore, increased for the whispered voice (not forced) from less than one foot to over twenty-five feet. The only evidence of the paralysis remaining is purely subjective, the patient stating that the left eye "waters" more easily than the right. In this period the patient has been seen and treated twenty-five times and the tube bougied three times.

It is beyond the scope of this paper to enter into a purely theoretical discussion of the possibilities which might be brought

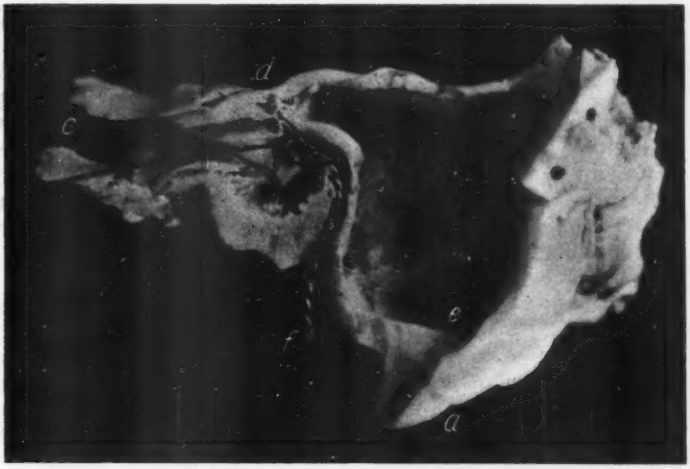
forward to explain this rare accident. The part played by individual predisposition and by the electricity in the production of the paralysis are questions too subtle for accurate determination. Such factors are, moreover, regarded as of little moment, even as contributing causes, in comparison with the mechanical forces, and the anatomical relations of the parts. A study of the gross anatomy of the Eustachian tube and the middle ear in fresh specimens and the skeletal preparations in the laboratory of the New York Eye and Ear Infirmary discloses anomalous conditions in a percentage of cases that can readily be made to explain this lesion.

In many fresh specimens the thin, curved lamina of bone forming the outer wall of the aqueductus Fallopii, situated above the fenestra ovalis and between it and the tegmen tympani on the inner wall of the tympanic cavity, is partially or entirely defective. This thin plate of bone is the only covering of the facial nerve as it passes through the inner wall of the tympanic cavity (second part of the nerve beyond the genu externum), except for the lining membrane of the tympanum and the nerve sheath. In quite a few of the specimens the ostium tympanicum was also found at an abnormally high level on the anterior wall of the tympanum (5 to 7 mm. instead of the normal 4 mm. above the lowest point on the floor). Thickening and unusual prominence of the upper part of the ostium (processus cochleariformis) was noted, which would obstruct the passage of the bougie into the tympanic cavity. The latter observation is of especial interest in connection with the studies of Bryant,[1] whereby it was demonstrated that strictures of the tube, when present, are more frequently met with at the openings of the tube than elsewhere. Such an obstruction yielding suddenly to pressure would allow the bougie to be advanced sharply and forcibly against the ridge of the aqueduct of Fallopius on the inner wall of the tympanum, provided that the ostium were abnormally high or that the aqueduct were unusually prominent in the second part of its course. Under normal conditions—as it can be demonstrated on anatomical specimens—the bougie on entering the tympanic cavity passes upward, outward, and backward at a very acute angle, or even parallel, to the ridge of the aqueductus Fallopii. Even when it impinges against the latter it is in the nature of a glancing blow. Given, however, a Eustachian tube actually shorter than the average (less than one and one-half inches), or admitting the tip of the silver catheter an eighth of an inch or more farther than usual, in connection with a fibrous or bony obstruction at the ostium tympanicum; or presupposing the latter opening to be at an abnormally high level; if the facial nerve is exposed by defective bony covering, with or without the dangerous proximity to

[1] Stricture of the Eustachian Tube in Aural Diseases and its Treatment, Annals of Otol., Rhin., and Laryng., June, 1905

the ostium produced by unusual prominence of the aqueductus Fallopii, the electro-bougie, on suddenly overcoming the obstruction at the ostium, may be driven against the facial nerve and bruise or crush it against the inner wall of the aqueduct, at a distance of one and one-half inches or more from the pharyngeal opening of the tube. In the accompanying illustration the bougie lies in the bony canal and strikes the facial (silk thread) at nearly a right angle.

The practial lesson of the case is obvious, even though the anatomical peculiarities cited are less frequent than would appear from the specimens studied. It is of great importance for the



a. Mastoid process. *b*. Bougie in the bony portion of Eustachian tube (part of wall removed by section). *c*. Carotid canal. *d*. Tegmen tympani. *e*. Mastoid cavity (cells removed). . Silk thread representing the course of the facial nerve through the aqueductus Fallopii.

physician and patient to be prepared for such a contingency, the bearing of which is immediate with reference to proper warning to the patient and careful technique, and remote in regard to serious permanent damage and ensuing medico-legal complications. In view, however, of the favorable prognosis and rarity of the lesion, and the great benefit to be derived and confidently expected, in the majority of such cases of impaired hearing, from the proper use of the electro-bougie, the possibility of such an accident does not form a serious objection, in my opinion, to this method of Eustachian dilatation.

CLINICAL AND EXPERIMENTAL EXPERIENCE WITH COLLOIDAL SILVER AND A VIRULENT STREPTOCOCCUS.[1]

By Frank P. Vale, M.D.,
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Any means by which one can cope successfully with a severe streptococcic infection, or prevent it, is of interest alike to the internist, surgeon, and obstetrician, because of the ubiquity of this organism, its protean manifestations, and its death-dealing properties. The internist now knows it is responsible for many cases of pleurisy, meningitis, acute articular rheumatism, suppurative arthritis, double parotiditis, hematuria, bronchopneumonia, enterocolitis, etc., though not always able to distinguish clinically those instances in which it is the etiological factor. To the surgeon and obstetrician it is only too well known as playing a large part in all wound complications, but fortunately their aseptic methods have curtailed its ravages. Colloidal silver is a remedy which has received much attention during the past three or four years. An impartial perusal of its literature cannot fail to impress one with its value in various infections, not only those due to the streptococcus, but to the staphylococcus, pneumococcus, anthrax bacillus, etc.

An allotropic form of silver was first described by Carey Lea in 1880,[2] but remained simply a laboratory curiosity until 1897, when introduced into surgical practice by Credé, of Dresden, in a paper read before the Twelfth International Medical Congress at Moskow. Credé had been lead to an inquiry into the therapeutic value of silver and its salts through observation of his father's well-known predilection for nitrate of silver in the treatment of ophthalmia, and also by his early recognition of the shortcomings of iodoform and mercurial preparations in wound treatment.

Nitrate of silver proved entirely unsatisfactory in the treatment of wounds, because of its ready decomposition and caustic action. Various albuminates were then tried, but his investigations were without result until Halsted's method of dressing wounds with silver-foil was witnessed. Following up this cue he found that metallic silver placed in aseptic wounds remained unchanged and was non-irritating, but when covering septic wounds, or a culture of one of the pyogenic cocci, the silver-foil disappeared, was dissolved—and in the Petri dish the silver was surrounded by a sterile zone. The only explanation was that the products of bacterial growth built up soluble antiseptic combinations with the silver. Chemical examination of the sterile zone surrounding the silver-foil in the culture

[1] Read at a meeting of the Medical Society of the District of Columbia, Washington, April 11, 1906.

[2] Silliman's Journal of Science, third series, vol. xxxvi, p. 477.

medium corroborated this hypothesis and demonstrated the lactate to be the principal compound thus formed. Experiments with the lactate and about twenty other organic salts of silver finally proved the citrate the most suitable for surgical purposes, and since then Credé has employed the citrate of silver with extreme satisfaction in the treatment of all wounds—for irrigation, impregnation of dressings, and for the sterilization of catgut.

As the result of this success the idea suggested itself that, as under the influence of pyogenic cocci antiseptic salts were formed from silver, perhaps it would be possible to bring about a disinfection, so to speak, of the entire body, if silver could be brought directly into the blood and body fluids, which would, of course, necessitate a form of metallic silver soluble therein. After consultation with many competent chemists, von Heyden produced a colloidal silver which, though not perfectly soluble, was applicable in following up this idea.

This preparation consists of small black particles of metallic luster, easily crushed, and containing 92 to 98 per cent. of silver; it is inodorous and almost tasteless. It is soluble in one to twenty parts of water; strictly speaking, however, it is not a solution, for the silver is merely in suspension, in particles so fine as hardly to be seen with the microscope; it filters through paper, but not through animal membranes.

At first it was employed chiefly in the form of a 15 per cent. ointment by inunction. It was rather irritating for hypodermic use. In the presence of a 1 per cent. egg albumen the silver is not precipitated by salts and acids, and in this combination has been administered by the mouth. In exceptionally severe cases Credé thought it might be given intravenously as bichloride of mercury was then being used by Bacelli, though at that time he had not thus employed it. Its intravenous use was taken up first at the Veterinary High School, Berlin, by Prof. Dieckerhof, and later by other veterinarians, and thus was successfully employed in the treatment of sepsis and various infectious diseases of cattle. Feeling thus relieved of the necessity of animal experimentation by its success in veterinary practice, Credé has employed colloidal silver by intravenous injection, since 1898, in the treatment of sepsis, pyemia, acute articular rheumatism, advanced pulmonary tuberculosis, and many other conditions, in doses of 5 to 20 c.c. of a 0.5 to 1 per cent. solution.[1]

My interest in colloidal silver, its value and limitations, especially when given intravenously, was aroused by the results noted in a most serious streptococcic infection seen in 1905. I had used it by inunction in the previous several years in a variety of affections, among them instances of streptococcic infection, and uniformly

[1] Berl. klin. Woch., September 16, 1901, p. 941.

with gratifying success, so I that was unprepared for the temporary failure which attended my efforts in this case.

On July 5, I was called to see a young man, aged nineteen years. For two days he had been feeling a little below par. His nose was slightly swollen, but that fact was not complained of, nor had it been scarcely noticed; his temperature was 102°. The condition was readily recognized as a facial erysipelas. I ordered the liberal application of Credé ointment to the nose and face, fully expecting, and in fact confidently predicting, a prompt cure. Perhaps this might have been the result had it not been for his indiscretion in getting out of bed the next morning, as he felt perfectly well. About three hours later I was hurriedly sent for, and found him suffering with a violent epigastric pain. The abdomen was quite soft, and my attention was immediately directed to the chest by his sighing respirations and tumultuous heart action. The lungs were normally resonant, but there was a pleuritic friction rub below the right nipple, and a blowing murmur at the apex of the heart; temperature, 104°. The streptococcus was evidently exercising its predilection for serous surfaces, as shown by the beginning pleurisy and endocarditis. An ice-bag was placed on the precordium, and during the next forty-eight hours Credé ointment was applied freely by inunction over the entire chest and abdomen.

On the evening of the third day it was apparent the infection was gaining the upper hand: an effusion had developed in the right pleural cavity; the heart action was more tumultuous, the blowing murmur at the apex louder; temperature, 104.6°. During the next twenty-four hours he was given five intravenous injections of 5 c.c. each of a 1 per cent. solution of colloidal silver, and one tub bath, at 80° F., of twelve minutes' duration: with the result that the apical murmur disappeared entirely, the heart action greatly improved, and the pleuritic exudation diminished considerably.

But at 10 P.M. on the fourth day, in spite of one-fourth gram of colloidal silver thus administered intravenously, his temperature was 105°; pulse, 100; respirations, 34; over the entire posterior aspect of both lungs were crepitant rales, impaired resonance, and increased vocal fremitus. Still more disheartening, not only had the swelling of the nose extended to the cheeks and scalp, but an entirely new focus of erysipelas, an area the size of the palm of the hand, had appeared on the calf of the left leg, at the site of an insect bite received in the woods a few days before the beginning of his illness. Extending from this new area up the leg to the knee were three parallel red streaks, looking like scratch marks, and indicating the course of inflamed lymphatic channels. A second bath of the same duration and temperature was given and a sixth intravenous injection, this time 10 c.c. of a 1 per cent. solution.

The morning of the fifth day the temperature dropped to 103.2°, respirations to 25, pulse to 80; the lungs were normally resonant

posteriorly where there had been beginning consolidation the night before. But at 4 P.M. the temperature was 105° in spite of the subsidence of the alarming chest symptoms; the new erysipelatous area on the leg had greatly increased in size, the entire leg was swollen, and the lymphangitis had extended to the groin; the urine was loaded with albumin and contained a few granular casts. The intravenous injection of colloidal silver was increased to 5 c.c. of a 5 per cent. solution, this amount being repeated at midnight and twice the next day. That ended the intravenous injections, for every available vein had been used until none could be entered again with the needle, by reason of considerable swelling of the arms in consequence of the escape into the tissues of some of the solution during attempts at injection into the smaller veins.

During ninety-six hours the patient received 1.5 grams of colloidal silver intravenously, 1 gram being injected during the last twenty-four hours of that period. The backbone of the attack was broken by this large dose of 1 gram intravenously inside of twenty-four hours, for on the evening of the sixth day, at the time of the last intravenous injection, the temperature had dropped to 102.2°, and the condition of the leg was rapidly improving. Had this large dose been employed from the start, or had I been able to continue it a short time longer, I believe this would have been the end of the attack.

The inunctions were continued, the face, entire front of the chest and abdomen, and the entire left leg being continually covered with the ointment and enveloped with oiled silk, as much as thirty grams a day being thus applied; this I supposed, of course, would be sufficient to maintain the good effects obtained by the intravenous injections. But in this I was to be disappointed, for on the evening of the eighth day, forty-eight hours after the last intravenous injection, the temperature again showed a tendency to rise, and the erysipelatous areas again began to extend.

Rectal injections of colloidal silver were resorted to, but did not control the smouldering embers of the infection until the dose was increased from 0.5 gram to 2 grams three times a day. For the next ten days, that is from the eighth to the eighteenth day of the disease, the temperature continued between 102° and 103°; but the morning of the eighteenth day it was so near normal that the rectal injection was reduced to 1 gram, and so continued until the twenty-first day, when symptoms appeared which indicated the limit had been reached in the administration of colloidal silver. Fortunately, it was no longer needed, for on the evening of the twentieth day the temperature was for the first time normal. By the rectum the patient had received 50 grams of colloidal silver; by the skin it is impossible to say how much was absorbed, but about 500 grams of a 15 per cent. ointment was used.

During the thirteen days of rectal injections the erysipelas cleared

on the face, after extending to the ears and well into the scalp. When the face had appeared quite normal for several days, the application of the ointment to the face was discontinued. Within forty-eight hours the lips and nose again began to swell, and though the inflammatory process was much less marked than in the primary attack, a dusky redness spread not only over the entire scalp, but this time down the neck, over the entire back and left arm, over the right shoulder and entire front of the chest, with here and there an angry patch the size of the palm of the hand. During this same period of rectal injections the focus on the leg extended below to the dorsum of the foot, where the swelling and inflammation were particularly severe, and above to the middle of the thigh, so that the only part of the entire cutaneous surface not involved at one time or another was the right arm and leg, the upper part of the left thigh, the buttocks, and in front the abdomen. It was found that after the migrating inflammation had passed a given point, and though for several days the skin had appeared normal, the inflammatory process would again flare up if the local silver application was discontinued, so that at one time about two-thirds of the body was covered with the ointment. In fact, after the temperature had been normal for a week, that is nearly a month from the beginning of the disease, the ointment was again left off the face, and again for the third time the lips and chin began to swell, and coincidently a new area of erysipelas appeared over the right trochanter; but both areas quickly subsided on the application of the ointment, and the temperature did not rise.

I was constantly on the lookout for some symptom indicating the limit had been reached in the administration of these large amounts of silver. The sclerotics had a peculiar blue tint suggesting the presence of a silver deposit in all the tissues. On the nineteenth day of the disease some pain in the stomach was complained of; and on the twentieth there was considerable vomiting of a watery fluid of such a gray tint as to suggest the presence of metallic silver, though none had been taken by the mouth. A specimen of the vomitus was examined by Dr. E. R. Hodge and found to contain silver. The gastric pain and vomiting soon disappeared on stopping the rectal injections.

The boy's troubles were not all over with the disappearance of the fever. During the next three weeks, that is from the twenty-first to the fortieth day, thirteen large abscesses formed in the left leg and arm, from which about two liters of pus was evacuated, containing as it was ascertained, the streptococcus in pure culture. In the left foot the inflammatory process reached almost the stage of gangrene; a four-inch incision on the dorsum disclosed a subcutaneous slough the size of the hand, and a second incision of two inches along the tendo Achillis showed a black necrotic area the size of a silver dollar. Most of the abscesses formed very rapidly,

practically with no local reaction; one or two hundred cubic centimeters of pus would be evacuated and the abscess cavity would close up in a most remarkable manner in three or four dressings. Two months after the subsidence of the fever, and when convalescence was nearly complete, the virulent streptococcus gave him a parting stab in the form of a small axillary abscess. It is needless to say his convalescence was very tedious; he was far more emaciated than any typhoid patient I have ever seen.

On the twenty-second day of the disease I was called about three o'clock in the morning, and found the patient in a state of collapse which nearly proved fatal; temperature 95° per rectum, breath and extremities cold, no pulse, and heart action barely audible. Hot salt solution per rectum with adrenalin brought a prompt response, though it had to be repeated several times to meet a recurrence of these symptoms. This collapse was evidently precipitated by the loss of fluids to the tissues incident to the copious vomiting of the previous twenty-four hours; and for a week fluids had to be administered freely to prevent a threatened recurrence.

The left arm could not be used for weeks. At one time it was so thin, the skin so tightly drawn over it, the biceps muscle so indurated, as to remind one of a well-dried part in the dissecting-room.

That the colloidal silver was to be credited with the cure of this patient was the unavoidable impression made upon me in watching it. Locally applied the silver exerted a most important local influence, for repeatedly when its local use was discontinued the inflammatory process in the skin flared up in areas which had apparently recovered completely some time before. The rectal injection, increased to 9 grams a day, finally overcame the remnants of the infection, but only after the backbone of the disease had been broken by the intravenous injection of 1 gram within a space of twenty-four hours—0.5 gram intravenously, extended over a period of seventy-two hours, having proved ineffectual.

Accumulated experience has proved conclusively the value of colloidal silver in combating various infections, both in the lower animals and in human practice, but it would extend this paper far beyond reasonable limits to attempt to review it. I now desire to turn to another phase of the application of colloidal silver, that is the question of its prophylactic value, concerning which there is but meager clinical and experimental data. Having in my possession, from this case, a virulent streptococcus which had been successfully combated, as just related, I undertook a few experiments on rabbits, with a view to obtaining further impressions as to its value and limitations in preventing infection by this same organism.

It was most natural for Credé to endeavor to extend the application of colloidal silver to the prevention of infection.[1] In recom-

[1] Monatsschr. f. Geburtshülfe und Gynäkologie, December, 1898, p. 583.

mending the insertion of suitable suppositories in the uterus and vagina after childbirth, he was simply making use of its local bactericidal effect. That was undoubtedly also his idea in leaving two or four tablets in the abdominal cavity after intestinal resection and similar operations especially likely to be followed by infection; the tablets were composed of 0.05 gram of colloidal silver rubbed up with a like amount of sugar of milk, so as to be readily soluble, and as many as ten tablets, he thought, might thus be allowed to remain in the peritoneal cavity. In fact, however, in addition to the bactericidal effect of the silver, he was unwittingly taking advantage of a property of the peritoneum which has only recently been the subject of laboratory inquiry, and hardly yet applied clinically: I refer to the local immunity acquired by the peritoneum against infection as the result of a passing irritation, it having been found that intraperitoneal injections of salt solution, urine, (2 per cent.), nucleic acid, boullion, blood serum, etc., so increases the resisting powers of the peritoneum that many times a lethal dose of various micro-organisms can be injected into its cavity, twenty-four hours later, without any effect.[1]

A further natural extension of these efforts at prophylaxis, and the outcome of his success in treating various infections, once established, was Credé's advice to give colloidal silver intravenously, during or following all septic operations, just as many surgeons give a saline transfusion.[2] In support of this departure he might have invoked the experience of Ewers in veterinary practice, though unique, I believe, in preventing by intravenous injection of colloidal silver a fatal dysentery which is endemic on some farms and attacks calves in the first days of life. The only experimental evidence I have been able to find favorable to colloidal silver is that of Beyer,[3] an assistant of Credé, which, however, is along the line of clinical and veterinary experience as to its value in combatting various infections once established, though in itself not conclusive, as but two dogs were used, and no control.[4] The only other experiments bearing in any way on the value of colloidal silver, either in combating or preventing infection, in which the small laboratory animals were employed, are those of Brünner, Trommsdorf and Cohn—all equally unsuccessful in demonstrating any virtue in prophylactic

[1] Issaeff, Zeit. f. Hygiene, 1894, p. 287; Borchard, Deut. med. Woch., 1904, No. 49; Miyake, Mitteilungen aus den Grenzgebieten der Med. u. Chir., 1904, xiii, p. 179; Petit, Ann. de l'Inst. Pasteur, 1904, xviii, 407; Durham, Medico-Chir. Trans., London, 1897, lxxx, 191; Mikulicz, Lancet, July 2, 1904, p. 1.

[2] Archiv. f. klin. Chir., 1903, xlix, 225.

[3] Münch. med. Woch., 1902, No. 8, p. 331.

[4] One dog was given intravenously 10 c.c. of a virulent staphylococcus culture. Twenty-eight hours later the animal was getting progressively worse; it then received 0.4 gram of colloidal silver, with prompt recovery, the temperature reaching normal on the fifth day. In a second dog he obtained the same favorable result from a similar dose subcutaneously, but the fever lasted ten days and the effect of the treatment was not so striking.

injections of colloidal silver against inoculation with various micro-organisms.

The article of Brünner[1] is much quoted, but only four of his experiments concern this aspect of the subject. In only one of them was the result favorable to colloidal silver: 1.50 of a staphylococcus culture was given a small dog intravenously, and a half hour later 0.1 gram of colloidal silver—the animal living seven and one-half days, while the control lived but eighteen hours. But in a second experiment a larger amount of the staphylococcus culture was injected and the animal died in seven and one-half hours, while the control lived. In the two remaining experiments the animals receiving the colloidal silver died as soon as the controls.

In the experiments of Trommsdorf[2] twelve dogs were given intravenous injections of colloidal silver varying from 0.02 to 0.03 gram, which in proportion to weight is about ten times the amount recommended by Credé as a prophylactic in human practice. Six, twelve, twenty-four, and thirty-six hours later a culture of the organism of swine erysipelas was injected intravenously—all the animals dying as soon as the controls.

The experiments of Cohn[3] are the most exhaustive. In two of them the cholera spirillum was the infecting organism: a guinea-pig receiving an intraperitoneal injection of a bouillon culture, followed by 0.05 gram of colloidal silver, lived four hours longer than the control; but a second guinea-pig died in twenty-four hours and the control lived. In four experiments with anthrax injected intravenously, followed by colloidal silver in dose of about 0.1 gram per kilo of animal weight, all the controls lived the longer. In two of four experiments with the staphylococcus injected intravenously the results were slightly in favor of the animals receiving the colloidal silver.

It will be observed that in these experiments, with the exception of two intraperitoneal inoculations, the infecting organism was thrown directly into the circulation, in relatively overwhelming numbers. This method of inoculation in no way simulates the manner of acquiring disease through the ordinary channels of infection, when the invading organism gains access to the circulation late in the course of the disease, or frequently not at all. In experiments with the streptococcus, however, three in number, Cohn inoculated the scarified ear of a dog with a loopful of the culture, followed by an intravenous injection of colloidal silver, about 0.1 gram per kilo of animal weight, and in each instance the control lived the longer.

But in these last mentioned experiments of Cohn the colloidal silver was injected at the time of the inoculation with the strepto-

[1] Fortschr. d. Medizin, May 16, 1900, p. 381.
[2] Münch. med. Woch., August 5, 1902, p. 1300.
[3] Centralb. f. Bacteriologie, 1902, vol. xxxii, p. 732.

coccus. Now the leukocytosis following the administration of colloidal silver, which is at its height at the end of twenty-four hours, plays an important, but as yet imperfectly understood, part in its action. It has been shown[1] in efforts at the prevention of infection through a hyperleukocytosis induced by the injection of nuclein, peptones, albumoses, and other substances, that to obtain favorable results it is necessary that the inoculation be made at the height of the induced leukocytosis. It was with the idea of filling up this small lacuna that I undertook the experiments mentioned, inoculating twenty rabbits subcutaneously twenty-four hours after the injection of colloidal silver, when the leukocytosis was at its height.

Twenty-four hours after an intravenous injection of colloidal silver the average leukocytosis was 12,000, due chiefly to an increase in the polynuclear cells; after intraperitoneal injections the leukocytosis was of about the same intensity, but due chiefly to an increase in the lymphocytes, as if the entire army of polynuclear phagocytes had responded to the local call in the peritoneal cavity. The degree of leukocytosis bore no relation to the amount of colloidal silver injected.

The streptococcus had been kept for a month without transplantation, yet it was found that 5 c.c. of a twenty-four-hour culture killed two-months'-old rabbits in about eighteen hours, when the inoculation was intravenous. The organism was peculiarly capable of surviving unfavorable conditions for growth, as it was successfully transplanted from an almost dry agar tube at the end of three months.

The colloidal silver was administered in some instances subcutaneously, and in others into the peritoneal cavity or intravenously, the dose varying from 0.003 to 0.500 gram per kilo of animal weight, in 0.5 to 5 per cent. solution.[2] The result of these prophylactic injections can be told in a few words. In no instance was a local reaction from the subcutaneous inoculation with the streptococcus entirely prevented, but with the largest dose of 0.5 gram per kilo of animal weight the inflammatory process was transitory and in marked contrast to that excited in the control. As the result of a particularly severe local reaction in a rabbit receiving intravenously 0.003 gram per kilo, I had my attention directed to the possibility of these smaller doses lessening the animal's resisting powers. In support of this idea it was repeatedly noted that the inflammatory reaction was much greater in animals given a previous small intra-

[1] Labbé, La Presse medicale, July 18, 1903, p. 519.

[2] In one rabbit the largest dose of 7 c.c. of a 5 per cent. solution was fatal when injected into a vein as rapidly as the syringe could be emptied. There were two or three convulsive movements, respirations ceased, the heart action continuing about three minutes. Postmortem: the left ventricle was found empty, while the right side of the heart and all the large veins were distended with a continuous blood-clot; the lungs were compressed against the back of the thorax, dark in color, and perfectly dry on section. The rapidity of the injection was a factor in producing this death, for a similar injection in another rabbit, given very slowly, was apparently without ill effect.

peritoneal injection of colloidal silver. Moreover, one rabbit was so far immunized by repeated small doses of the streptococcus as twice to have no local reaction, but when again inoculated twenty-four hours after a subcutaneous injection of colloidal silver, 0.004 gram per kilo, the usual erysipelatous inflammation appeared. All the controls lived, while two of the animals receiving small doses of colloidal silver and inoculated as related, twenty-four hours later with the streptococcus, died: in one, the dose was 0.070 gram per kilo subcutaneously, and in the other, 0.030 gram per kilo intravenously.

Of course, these experiments do not permit of any broad generalizations, but taken in connection with those of Brünner, Trommsdorf, and Cohn, they at least suggest—in spite of the well-demonstrated value of colloidal silver against infection once established—that in doses heretofore advocated, no confidence can be placed in its ability to prevent infection; and they further suggest these small doses may even lessen the patient's resisting powers, whether used intravenously, in the peritoneal cavity, or subcutaneously, and actually be an added source of danger.

A NOTE ON THE PRODUCTION OF VASCULAR LESIONS IN THE RABBIT BY SINGLE INJECTIONS OF ADRENALIN.[1]

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The many publications which have followed Josué's demonstration of the power of adrenalin to produce degenerative changes in the rabbit's aorta have added much to our knowledge of the degenerative, infiltrative, and reparative processes which may occur in bloodvessels; but, unfortunately, from the point of view of comparative pathology, the value of this method of experimentation has been lessened by the failure to produce lesions entirely analogous to those of arteriosclerosis in man—the condition which it most closely resembles, and further, by the absence of an adequate explanation of the manner in which adrenalin acts to produce such lesions.

[1] Read at the meeting of the Association of American Physicians, Washington, D. C., May 15 and 16, 1906. This work was conducted under a grant from the Rockefeller Institute for Medical Research.

This method, however, is so full of promise that it seemed advisable to continue the investigations[1] carried on in this laboratory during the past two years, in the hope of producing, by varying the conditions of the experiment, lesions more closely resembling those of arteriosclerosis in man, and at the same time to study by exact physiological methods the action of adrenalin. These studies are as yet incomplete, and it is our intention in this communication to refer only to the lesions caused by single injections of adrenalin.

That repeated frequent injections of adrenalin are necessary to the production of experimental vascular lesions appears to be the opinion of all who have used it. Loeb and Githens[2] have stated that lapse of time is of more importance than frequency of injections or size of dose, and although they alone appear to have noted this fact they did not materially limit the number of injections in their experiments.

Our attempt to produce lesions by single injections was mainly for the purpose of testing the theory offered by Pearce and Stanton, and later supported by the former in his study of experimental myocarditis due to adrenalin. This was, briefly, that in the aorta and the myocardium the primary condition is a localized anemia due to the direct action of adrenalin on the arterioles of areas not readily supplied by collateral circulation. To this factor is added the influence of the strain thrown upon these poorly nourished parts during the height of the increase in blood pressure, an influence sufficient presumably to cause permanent injury. If lesions could be produced by single injections of adrenalin this theory would be strengthened and that of direct toxic action weakened.

As it had been previously shown that necrotic foci were present in the aortas of animals that had received four and five injections, three groups of animals were given respectively one, two, and three intravenous injections of one-half to one and a half minims of a 1 to 1000 solution of adrenalin (Parke, Davis & Co.). Although lesions were produced in animals that had received two and three injections, they became of little importance, in view of the very positive results following single injections. Of ten animals in the latter series, four died within a few minutes after injection; of the remaining six, one of which died at the end of seven days, and five of which were killed after sixty-five to seventy days, all showed very definite vascular lesions. The protocols of these experiments may be briefly summarized as follows:

Experiment 13. A full-grown rabbit received in the ear vein on December 9, one-half minim of a 1 to 1000 solution of adrenalin.

[1] R. M. Pearce, and E. McD. Stanton, Experimental Arteriosclerosis, Jour. Exper. Med., 1906, viii, 74; R. M. Pearce, Experimental Myocarditis: A Study of the Histological Changes following the Intravenous Injection of Adrenalin, ibid., 1906, viii, 400.

[2] The Effect of Experimental Conditions in the Vascular Lesions Produced by Adrenalin, Amer. Jour. Med. Sci., 1905, cxxx, 658.

No ill-effect was noticed after recovery from the initial disturbance. On December 16 the animal was found dead. Postmortem examination revealed œdema of the subcutaneous tissues, minute hemorrhages in the intestinal serosa, and about 20 c.c. of slightly blood-tinged fluid in the peritoneal cavity, but no increase of fluid in the pericardial or pleural cavities. The heart was somewhat dilated and its muscle pale. Beneath the epicardium were numerous punctate hemorrhages. The aorta throughout the thoracic and abdominal portions presented a peculiar wrinkled appearance without well-defined thickening or change in color. The other organs were not noteworthy.

Histological examination of the aorta at different levels revealed an extensive and diffuse degeneration of the media with more or less complete necrosis of the muscle cells, and a straightening and fusion of the elastic fibers with a few angular bends. About two-thirds of the circumference of the vessel were involved and transversely the middle third of the media. In the heart muscle were focal areas of degeneration, with slight increase of connective tissue and accumulation of lymphoid cells.

Experiment 17. A rabbit weighing 1100 grams was injected on January 4 with one-half minim of adrenalin. It was chloroformed on March 14; weight 1710 grams. Upon postmortem examination 75 c.c. of clear watery fluid was found in the peritoneal cavity. The aorta from its origin to the cœliac axis presented numerous irregular calcified plaques varying in size from 0.2 to 1.5 cm. in diameter. The other organs showed no changes. Upon histological examination the aortic lesions were those of degeneration and calcification.

Experiment 20. A rabbit weighing 1920 grams was injected on January 9 with one minim of adrenalin. Killed March 14; weight, 2120. Postmortem examination revealed no lesions except in the aorta, which from the origin to the bifurcation was dotted with oval and linear elevated areas of thickening and calcification. The areas were larger and more abundant in the ascending portion. Well-marked thickening was present about the orifices of the coronary arteries. Histological examination showed the usual picture of necrosis and calcification.

Experiment 21. A rabbit weighing 2230 grams received on January 9 one and one-half minims of adrenalin; it was chloroformed on March 15. At autopsy no changes were found except in the aorta, which presented just above the cœliac axis a single irregular area of calcification 0.8 cm. in diameter. Upon histological examination very extensive repair was evident about the calcified area, especially in the subintimal tissues.

Experiment 22. A rabbit weighing 1860 grams was injected on January 9 with one and one-half minims of adrenalin. It was chloroformed on March 15; weight, 1910 grams. In the aorta just above the cœliac axis were two calcified patches, the larger of which

measured 0.5 cm. in diameter. The histological picture was that of calcification, with no repair.

Experiment 23. A rabbit, 1950 grams in weight, was injected on January 9. It was killed March 14; weight, 2700 grams. In the upper portion of the abdominal aorta was one small wrinkled thickening 0.3 mm. in diameter, which presents histologically the picture of calcification.

These results, while they offer no explanation of the mode of action of adrenalin in producing vascular lesions, simplify the mode of investigation, in that the problem is limited to the study of the effect of single injections, and comparisons may be drawn sharply between lesions so produced and similar lesions caused by the prolonged administration of other substances, such as nicotin, digalen, and barium chloride. It is evident, however, that in view of these results the theory of direct toxic action is hardly tenable, while that which recognizes a combination of vasovasorum disturbance, local anemia, and vascular strain is greatly strengthened.

BACILLUS PYROGENES (NOV. SPEC.?) ASSOCIATED WITH A FEBRILE DISEASE.

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The following account of an unusual disease with the pigmented bacillus isolated from it, is published on account of its general interest and the impossibility of identifying the organism associated with it. The descriptions of most pigmented bacteria are so meager and faulty, especially the absence of animal experiments, that it is dangerous at this day to announce a new species. I even hesitate to go so far as to conclude from the evidence that the bacillus is the etiological factor and responsible for the disease. Yet it seems from the tissue changes in inoculated animals that this bacillus produces a characteristic, well-definable pathological condition.

A search through the literature has yielded no evidence of an organism described completely enough to compare fully with Bacillus pyrogenes (nov. spec.?). This name has been selected at the suggestion of Professor William H. Welch, to whom I am much indebted.

On January 2, 1906, a gentleman thirty-six years of age, who had a slight fever, was sent from Lynchburg, Virginia, to Prof. George J. Preston, of Baltimore. The patient was taken to the Baltimore City Hospital, suffering with a number of nervous symptoms, and

slight fever; his temperature ranged between 99° and 100° and 101°, and reached 102° F. only once. His most striking symptoms were meningeal: photophobia, hyperesthesia, restlessness, tremors, both coarse and fine; Kernig's sign was present, and various joints were swollen and painful at different times.

An examination of the patient's blood for typhoid bacilli was negative. A blood count showed a leukocytosis of about 14,000. The patient had a typhoid appearance, but had no rose spots or enlarged spleen. The most striking feature of his symptoms was the involvement of the joints; as one subsided another flared up. The patient refused permission to take cultures from the joints. Prof. Preston felt convinced, from the start, that the case was not one of infection from typhoid bacilli. On February 20, the patient still had fever, whereupon Prof. Preston kindly asked me to make a blood culture. The patient was finally discharged on March 15, 1906. He has remained perfectly well since.

During the patient's presence in the hospital it was possible to make one only agglutination test with the organism which was isolated from his blood. At the end of two hours no agglutination had taken place.

The three Ehrlenmeyer flasks into which the 10 c.c. of blood had been distributed were incubated at a temperature of 37° C., for seventy-two hours. Colonies grew from only one flask.

The surface colonies after twenty-four hours showed themselves on the average about the size of a pin-head. They were small, round, and lemon-colored, and varied in shape from a perfect circle to that of an oval; colonies were slightly raised, shiny, somewhat translucent, and moist. By an inverted No. 3 eye-piece they were yellow, finely granular, with more or less regular edges. The deeper colonies were more concentrated, smaller, and contained a darker nucleus.

On slant agar the growth remained confined to the line of the needle; the stroke was a very deep lemon-yellow color. It was diffuse, irregularly filiform, moist, slightly raised, and shiny.

After seventy-two hours the growth seemed to have reached its maximum. At this time, compared to a seventy-two-hour-old typhoid culture, it was about twice its thickness and twice its width.

Cover-glass preparations contained a small diplobacillus, with rounded ends and almost as broad as long. It measured from 0.6 to 1.4 μ long by 0.5 to 0.8 μ thick. It was completely discolorized by Gram's stain. No spores were found. Capsules were present. It was a flagellated organism of the peritrichal variety.

Bouillon had faint cloudiness. Loops of bouillon in hanging-drop preparations showed very actively motile bacilli in pairs and short chains. The motility was equal to or exceeded that of the typhoid bacillus.

In gelatin the puncture gave a very characteristic growth after

twenty-four hours. On the surface for a few days the growth was no larger than the width of the needle. After the fifth day the surface growth spread to a millimeter in diameter, and took on a deep-yellow color. In the depth of the gelatin, along the stab, the growth spread very slightly, became somewhat granular and filamentous, with a pale-yellow tint. In all of the gelatin stabs a beautiful spiral of discrete granular colonies began to appear at the bottom of the stab about the fourth day. Until the seventh day there was no sign of liquefaction, after which it liquefied rapidly.

The growth on potatoes was thick, opaque, and profuse; dark lemon-yellow in color, raised, with a velvety surface, shiny and dry.

Litmus milk became very slightly acid at the end of twenty-four hours. At the end of two weeks many small particles of coagulated casein were seen floating on the surface of the milk.

On peptone there was a cloudy, yellow growth.

No indol was formed.

Fermentation tubes, which contain lactose bouillon, glucose bouillon, or saccharose bouillon, when inoculated with Bacillus pyrogenes, produced no gas.

Litmus agar, lactose litmus agar, and mannite litmus agar were acidulated.

The thermal death-point of Bacillus pyrogenes, exposed for ten minutes, is 60°.

There was no odor, pleasant or unpleasant, attached to any of the cultures.

The pathogenic power of Bacillus pyrogenes was tested by inoculating four rabbits and two guinea-pigs intravenously, intraperitoneally, and subcutaneously. The two former methods, when employed in these experiments, were very effective in killing the animals. Animals inoculated subcutaneously did not die.

Four cubic centimeters of a twenty-four-hour bouillon culture of Bacillus pyrogenes was inoculated into the ear vein of a large gray rabbit at 11 A.M. February 27, 1906. It was alive and feeding, but not very active, at 3.30 P.M. At 4 P.M. it was found dead. The autopsy was performed at 12.30 P.M. February 28.

A second rabbit was inoculated with 4 c.c. of a twenty-four-hour old pure bouillon culture of Bacillus pyrogenes on February 27. Inoculation was made into the left hind knee or second joint. On February 28 a small amount of fluid seemed to be present and some capitus was felt; this remained until March 7, when the rabbit was found dead.

A third rabbit (small albino) was inoculated in the ear vein on March 1, at 2 P.M., with 0.5 c.c. of a pure bouillon culture of Bacillus pyrogenes. The rabbit was killed on March 4. Pure cultures of the organism originally inoculated were obtained from the blood and all the viscera. A description of this autopsy tallies exactly with autopsy No. 1.

A large male guinea-pig inoculated in the peritoneal cavity with 1 c.c. of a twenty-four-hour bouillon culture of Bacillus pyrogenes on April 7, at 1 P.M.; the guinea-pig was found dead.

Pure cultures of Bacillus pyrogenes were obtained from all these autopsies. A complete description of them and of the tissues is reported in another place.

After searching the literature carefully I have been unable to find any organism which in the slightest degree resembles this one. Dr. Stokes called my attention to the description of an organism found in Manila, P. I., and reported by Lieutenant Maximilian Herzog. After carefully comparing these organisms, his and the one I report, I cannot find the slightest resemblance. His organism was obtained at an autopsy performed by him on an unknown native islander who was brought to the hospital dead. The patient's previous history was entirely unknown. At the autopsy the only lesions found were general glandular enlargements and an incised wound in the left leg. The liver, spleen, and kidney showed parenchymatous degeneration. The liver showed some interstitial hepatitis.

Cultures taken from the various organs, after twenty-four hours, showed no growth except that taken from the liver. The culture made from the liver grew an organism with a heavy golden-yellow pigment and exuding a fœtid cadaverous odor; hence, Dr. Herzog has called his organism (ignoring the binominal nomenclature) "Bacillus aureus fœtidus."

It will be easier, perhaps, to compare his organism with the one isolated by me from Prof. Preston's patient, by placing them side by side in the following table:

	Bacillus pyrogenes.	*Bacillus aureus fœtidus.*
Pigment	Lemon yellow	Golden yellow
Capsule	Absent	Present
Spores	Negative	Positive
Motility	Active	Absent
Size	0.6 micron to 2.1 microns	0.4 micron to 1.4 microns long
	0.5 micron to 0.8 micron	0.4 micron to 1.4 microns wide
Liquefaction	Complete in twelve hours	Absent until seventh day
Odor	None	Cadaverous
Death-point	60°	62°

To summarize, a male patient, aged thirty-six years, with a temperature lasting eight weeks, which never rose above 102°, exhibited meningeal symptoms, photophobia, tremors, irritability, subsultus tendinum, and joint involvement; the Widal reaction was constantly absent; there was a leukocytosis of 14,000. Cultures taken from his blood revealed a lemon-yellow diplobacillus pathogenic for guinea-pigs and rabbits, negative to Gram's stain, actively motile, producing no indol, not liquefying gelatin until the seventh day,

and producing no gas in lactose, saccharose, or glucose media. Milk, mannite, litmus agar were acidified. The organism was killed at an exposure of 60° C. for ten minutes. It has been named Bacillus pyrogenes.

In conclusion, I wish to extend thanks for kind suggestions and assistance to Prof. George J. Preston, through whose generosity I was enabled to do this work; to Prof. Wm. H. Welch, for naming the organism, and especially to Prof. Wm. Royal Stokes, for much assistance.

CERTAIN REMOTE CONSEQUENCES OF INFECTIONS OF THE BILIARY TRACT, WITH SPECIAL REFERENCE TO, (1) CHOLELITHIASIS AND CHOLECYSTITIS, (2) ADHESIONS OF THE UPPER ABDOMEN, (3) THE GENERAL PRINCIPLES OF TREATMENT, AND (4) THE INDICATIONS FOR SURGICAL INTERVENTION.[1]

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In discussing the more immediate consequences of infection of the biliary tract,[2] I mentioned that virulent bacteria and definite anatomical lesions may persist in the gall-bladder for years, and that such subacute or chronic, insidious, and long-standing infections play a major role in the etiology of gallstones, cholecystitis, pericholecystic adhesions, and the divers symptoms commonly but often erroneously ascribed to disorder of the stomach.

THE INFECTIOUS NATURE OF CHOLELITHIASIS, CHOLECYSTITIS, AND THEIR COMPLICATIONS.

In asking your attention now to a discussion of the infectious nature of gallstones and the entire cholelithitic process, it is not my intention to review in detail the manner of formation of gallstones; this was done some years ago by the master hand of Naunyn. But I may point out briefly the accepted opinions on the subject.

There is general agreement that the one necessary factor in the

[1] Part II of the Mütter Lecture of the College of Physicians of Philadelphia, delivered December 1, 1905. Part I of the Lecture, entitled "Infections of the Biliary Tract, with Special Reference to Latent (or Masked) and Typhoid Infections," was published in the Amer. Jour. Med. Sci., September, 1906, cxxxii, 446.

[2] Part I of this Lecture, loc. cit.

causation of gallstones is a low-grade catarrhal inflammation of the biliary tract; the second requisite seems to be some obstruction to the free flow of bile. The one without the other factor is not sufficient; whether a third factor is of significance remains to be determined. In consequence of bacterial infection of the biliary tract a low-grade catarrhal inflammation is set up; this, on the one hand, leads to obstruction to the free flow of bile from swelling of the mucous membrane, and the products of this inflammation, on the other hand, contain the essential constituents of gallstones. In the gall-bladder (the seat of formation of most gallstones) the catarrhal inflammation leads to desquamation of the lining epithelium, an albuminous exudation, and an increased formation of mucus and of cholesterin; and in the gall-bladder dynamic factors doubtless frequently favor the stagnation of bile. The increased cholesterin, then, is derived not from the bile (being according to Naunyn, independent of general bodily and diathetic conditions); it results from catarrhal disintegration of the mucous cells lining the wall of the gall-bladder. An early evidence of this increased formation of cholesterin may be seen microscopically in the so-called myelin droplets or bodies within the mucous cells. The second important constituent of the gallstones, bilirubin-calcium, is derived from the bile, being precipitated by the albuminous exudation of the inflammatory process—a phenomenon that can be imitated experimentally by adding egg albumen to normal bile. Stagnation and inspissation of the bile, to which, according to the older views, the formation of gallstones was due, do not give rise to the increased formation of cholesterin nor to the precipitation of bilirubin-calcium—whence they are contributing factors only. Bilirubin-calcium is believed by Naunyn to act as a cement substance, binding together the cholesterin, desquamated epithelium, etc.—the whole forming the nucleus of the gallstone. The exact significance of foreign bodies and of chemical substances apart from bacterial products has not yet been definitely determined. There being no cholesterin-bearing mucous membrane in the smaller bile ducts, cholesterin gallstones are not formed within the intrahepatic ducts, although by a retrograde movement they may be transported thither; bilirubin-calcium calculi only are formed in the intrahepatic ducts; but both varieties, as well as mixed calculi, are formed within the gall-bladder.

Whether infection and obstruction to the free flow of bile comprise all the necessary factors in the causation of gallstones remains to be definitely determined; but there is some reason for concurring with Herter[1] when he says: "It is plain from what has been said that there is at present no unequivocal evidence that gallstones arise from constitutional derangements unconnected with microorganismal invasions of the gall-bladder. On the other hand, it

[1] The Etiology and Chemical Pathology of Gallstones. Trans. Cong. Amer. Phys. and Surg., 1903, vi, 158.

is certain that the cholesterin of the bile can be considerably increased by local irritants unconnected with infection, and it is likely that the requisite local conditions for such increase sometimes arise through purely metabolic disorders. While gallstones are commonly the result of local infections, we should carefully guard against the conclusion that they can never have a diathetic origin. It is at least highly probable that diathetic conditions are capable of so altering the composition of the bile as to favor materially the production of calculi in the presence of suitable local bacterial activities."

Beer[1] also has recently suggested that a third factor which, he says, may be called altered liver metabolism or a diathesis, may be of significance in the etiology of gallstones. He believes that Naunyn's factors, stagnation of bile and inflammation of the biliary passages, do not seem to be sufficient in themselves to lead to gallstone formation, even though the time allowed for the working of the causes be adequate, and that these factors lead to gallstones only in persons who previously have had gallstones.

For the present, however, I am content to point out that in at least the very great majority, if not in all cases, gallstones have a micro-organismal origin, that they arise in consequence of infection of the biliary tract. What, however, is of even greater importance is the role that bacteria play in the secondary infections following the formation of gallstones.

The consequences of gallstone activity may be mechanical or inflammatory or both. The mechanical consequences follow what may be designated wandering of the stone into and through the biliary passages. In consequence of this activity, the gallstone may set up mechanical effects in the gall-bladder, in the cystic duct, in the hepatic duct, in the common bile duct, in the diverticulum of Vater, or in the intestine. These effects are of great variety and of varying importance. Probably the most important and serious comprise permanent complete obstruction of the common bile duct, with consequent permanent jaundice, dilatation of the bile ducts, and final biliary cirrhosis of the liver. Of only less importance are the dragging sensations and discomfort attendant upon the weight of many gallstones in a distended gall-bladder; the gradual production of a Riedel's or a linguiform lobe of the liver; the effects of pressure on adjacent organs; complete obstruction of the cystic duct which, as a rule, leads at first to some distention of the gall-bladder, but soon to absorption of the bile and its replacement by mucus (hydrops) and gradual shrinkage of the gall-bladder, etc. These and other mechanical consequences of gallstone activity, however, especially incomplete obstruction of the ducts, acquire their major importance from the opportunity that they afford for bacterial infection; in fact, they invite such infection, and the infection is usually a concomitant condition.

[1] Concerning the Causes of Gallstones, AMER. JOUR. MED. SCI., 1905, cxxx, 432.

This infection of the biliary tract is of the utmost significance, and forms an integral part of what is commonly designated calculous cholecystitis and cholangitis. The phenomena may develop (1) acutely, without preceding clinical signs referable to the gall-bladder; (2) subacutely, and (3) chronically. The ensuing pathological lesions are of the greatest diversity: Thus, for instance, gallstones may or may not be present, and the concomitant inflammatory phenomena may be of varying grades—from the mildest catarrhal lesions to widespread phlegmonous and ulcerative processes that may lead to perforation or gangrene of the gall-bladder; gallstones, if present, may be quiescent or active; they may be present in the gall-bladder, or in any one of the ducts, or in all the ducts, or in the ducts and not in the gall-bladder; they may cause an acute or chronic, partial or complete, temporary or permanent, obstruction of the cystic, the hepatic, or the common bile ducts, and, on the other hand, such obstruction may occur in the absence of gallstones (being due to swelling of the mucous membrane, kinking of the ducts, or obstruction from without), and in the presence of gallstones the ducts may be partially or completely patulous; the gall-bladder may be distended or contracted, its walls thinned or much thickened, and its lumen ultimately may become almost if not quite obliterated; it may contain bile, mucus, blood, or pus, or combinations of these, in addition to or in the absence of gallstones; adhesions may form between the gall-bladder and adjacent structures (the liver, the stomach, the duodenum, the colon, the omentum, etc.), and by way of the adhesions, the gallstones may rupture into the gastrointestinal tract and sometimes cause intestinal obstruction; or purulent pericholecystitis and pericholangitis, localized or generalized peritonitis, pylephlebitis, pericholangitic abscesses of the liver, fistulæ, acute and chronic pancreatitis, etc., may ensue; and finally, in some cases a general bacterial, often pyococcic, infection, with or without multiple abscesses, may develop.

All of these pathological lesions were well exemplified in the 216 cases under review. It is not pertinent at present to give a detailed statement of all the lesions, but reference to some of them may be of interest. Thus:

Of 216 patients, 182 (84.2 per cent.) had gallstones, and 34 (15.8 per cent.) had no gallstones.

Of 182 patients that had gallstones, 101 (55.5 per cent.) had stones in the gall-bladder alone; 23 (12.5 per cent.) had stones in the gall-bladder and cystic duct; 19 (10.5 per cent.) had stones in the gall-bladder and common duct; 12 (6.6 per cent.) had stones in the common duct alone; 11 (6.0 per cent.) had stones in the cystic duct alone; 5 (2.8 per cent.) had stones in the gall-bladder, cystic, hepatic, and common ducts; 4 (2.2 per cent.) had stones in the gall-bladder, hepatic, and common ducts; 1 (0.6 per cent.) had stones in the gall-bladder, cystic, and common ducts; 2 (1.1 per cent.) had

stones in the adhesions (not otherwise specified); and 4 (2.2 per cent.) had stones in the regions not definitely specified.

Of 216 patients, 58 had no stones in the gall-bladder, and of these, 23 had stones in the ducts. 41 had stones in the common duct, and of these, 12 had stones nowhere else. 9 had stones in the hepatic ducts (as well as elsewhere).

Of 216 patients, 123 (56.9 per cent.) had adhesions about the gall-bladder; 22 (10.1 per cent.) had no adhesions; and of 71 (33.0 per cent.) there is no note in the case-history of the presence or absence of adhesions (probably none were present).

The mechanical and the inflammatory phenomena attendant upon gallstones may occur concurrently or alternately, and they give rise in consequence to a most complex and variable symptomatology. It is altogether without the province of this lecture to discuss this symptomatology in detail, but several of the clinical manifestations of these remote consequences of infections of the biliary tract, as exemplified by the 216 patients under review, may be mentioned: (1) The pains; (2) the jaundice; (3) the size of the gall-bladder; and (4) the accompanying fever.

The Pains. Of 216 patients, 161 (74.5 per cent.) had colicky pains; 45 (20.8 per cent.) had non-colicky pains; 7 (3.3 per cent.) had pain not accurately described; and 3 (1.4 per cent.) had no pain whatever (one was markedly jaundiced, and the other two had fever, 100° and 104.6° F. respectively).

Of 161 patients that had colicky pains, 137 (85.1 per cent.) had gallstones, and 24 (14.9 per cent.) had no gallstones.

Of 45 patients that had non-colicky pains, 35 (77.7 per cent.) had gallstones, and 10 (22.3 per cent.) had no gallstones.

Of 3 patients that had no pains whatever, 2 had gallstones. 15 patients never had colicky pains or jaundice, and of these 12 had gallstones, and 3 had no gallstones.

In seeking a rational explanation of the pains of gallstones, several facts stand in relief: (1) That gallstones are present in very many subjects and are commonly believed not to cause noteworthy symptoms; (2) that symptoms having once occurred are exceedingly likely to recur; and (3) that symptoms are often paroxysmal and very severe. It is desirable for us to inquire, in the first place, whether it is really true that only a very small percentage of gallstone subjects (commonly estimated at 5 per cent.) manifest no noteworthy symptoms; and, in the second place, we have to explain why a quiescent stone becomes active—why a gall-bladder that for years has harbored a stone or stones suddenly occasions symptoms; and why the symptoms are often paroxysmal and very painful. As regards the presumed symptomless course of cholelithiasis in many subjects, I need only emphasize the fact now well known to surgeons, but less widely acknowledged by some general practitioners, that the congeries of symptoms commonly denominated "stomach trouble,"

"indigestion," etc., are due in many cases to disease of and about the biliary tract. In a number of cases in which gallstones are an accidental finding at the necropsy, investigation of the past history of the patient reveals many and often long-continued attacks of "indigestion" (epigastric distress, anorexia, flatulence, eructations, nausea, vomiting, etc.); the stomach is often normal—in which event, the gallstones and possibly pericholecystic adhesions, rather than being accidental, serve to explain the clinical symptoms. As practising physicians, it behooves us to be on the alert to recognize disease of the biliary tract in the absence of jaundice and gallstone colic, and we must renounce the tenet that gallstones in the great majority of cases cause no symptoms; they do cause symptoms—symptoms, however, referable often to the stomach rather than to the biliary tract.

As regards the pains of cholecystitis, the weight of evidence at hand strongly supports the opinion that an active infection of the biliary tract, that is, a cholecystitis or cholangitis, is the factor in provoking activity where quiescence formerly reigned. As is well known, the pains may be local or referred, or both, and the local pain may be colicky or non-colicky. Although there is some discussion as to the cause of the colicky pain, there is little or no objection to attributing the non-colicky pain to inflammatory phenomena provoked by infection, inflammation of and about the gall-bladder and biliary ducts. The dull aching pains are with excellent reason ascribed to inflammatory changes in the gall-bladder with the exudation of inflammatory products, and consequent distention; the more acute pains, associated with muscular rigidity, tenderness, etc., are due to concomitant infection of the regional peritoneum—which as it subsides occasions peritoneal adhesions, a contributing factor in the dull aching pains, and of other significance to be presently mentioned. We must bear in mind, however, that some considerable distention of the gall-bladder is possible without provoking noteworthy pain—provided the cystic and the common ducts are patulous. I have seen altogether painless, though much enlarged and somewhat tender, gall-bladders in the course of typhoid fever. It is to a similar cause, that is, efficient drainage, that the comparatively symptomless course of some cases of cholelithiasis is to be attributed, although the absence of inflammatory phenomena in the wall of the gall-bladder and the ducts, and the absence of pericholecystic adhesions, are also of significance. As regards the referred pains (epigastrium, subscapular region, the neck, the shoulder, the arm, etc.), although the sympathetic nerve is of significance, the demonstration that filaments of the pneumogastric nerve are distributed to the gall-bladder serves to explain the common association of gastric and cardiac symptoms with disease of the biliary tract; and the frequent occurrence of pain, sometimes of actual colic, three or four hours after a meal suggests a causal relationship to

the normal physiological contraction of the gall-bladder induced by the entrance of the chyme into the intestine.

As regards the colicky pains, I am firmly of the opinion that heretofore we have been too much concerned with interpreting them as evidence of the passage of a gallstone through the biliary ducts; pains of like character may doubtless be due to different factors—which have been well pointed out by Riedel, who gives the following as the causes of gallstone colic: (1) Adhesions of a gall-bladder no longer containing stones; (2) adhesions when large stones are present in the gall-bladder and the cystic duct is patent; (3) inflammatory processes in a gall-bladder distended by fluid or stones, when the cystic duct is occluded by inflammation or by the presence of a stone in the neck of the gall-bladder; (4) the transit of a stone through the bile passages; and (5) the inflammation of a dilated, calculous common duct, or its tributaries, without impaction of the stone.

There can be little doubt that in a number of cases the spasmodic efforts of the gall-bladder and the common duct to expel their contents by vigorous contraction of their muscular coats are the cause of the peculiar gallstone colic. This pain occurs in its greatest intensity during the transit of a stone through the ducts, and it ceases as the stone is discharged into the intestine or drops back into the gall-bladder—thus freeing the cystic duct. But there can also be little doubt that attacks of pain indistinguishable from those provoked by the transit of a stone may be due to other factors—those mentioned above; although in some instances the attacks of pain may not be as severe as those of a so-called true gallstone colic. This pain may be likened to that provoked by the efforts of other hollow viscera, such as, the appendix, the intestine, the ureter, the urinary bladder, the Fallopian tube, etc., the contractions of which designed to expel their contents against an obstruction are very painful; and, in the case of the gall-bladder, as well as of these other viscera, if the obstruction be incomplete, hypertrophy of the muscular coat results, and often later lessening of the size of the cavity, whereas, if the obstruction be complete, paresis of the muscular coat soon occurs, dilatation ensues, and the pain subsides. But, as exemplified by the 216 patients under review, gallstones are absent in 15.5 per cent. of the patients who have colicky pains, and they are present in 77.7 per cent. of the patients who have only non-colicky pains.

Furthermore, as is well known, gallstones may be passed by the bowel, that is, they may ulcerate into the bowel, forming a fistula, without colicky pains having ever been experienced by the patient. As regards the cause of the colic, I believe that, although mechanical factors, such as traumatism, joltings, etc., may sometimes be the immediately provoking factor, it results in the great majority of cases from infection and consequent inflammation; that is, cholecystitis provokes activity, as it were sets the stone in motion; and that

when no stone is present the increasing tension within the gall-bladder and the spasmodic efforts to empty itself cause the pain. We should, therefore, interpret the great majority of gallstone colics, not as evidence of the passage of a stone through the biliary ducts, but as evidence of an acute cholecystitis. A fact tending to support this view is that as the inflammation subsides the stones become quiescent; indeed, the one hope of medical treatment is to cause subsidence of the infection and inflammation.

THE JAUNDICE. Of 216 patients, 138 (63.9 per cent.) had jaundice at some time or other; 74 (34.2 per cent.) never had jaundice; and of 4 (1.9 per cent.) there is no statement as to the occurrence of jaundice. 54 (14.0 per cent.) had jaundice at operation and previous attacks; 13 (6.0 per cent.) had jaundice at operation and no previous attacks; 67 (31.0 per cent.) had previous attacks of jaundice but no jaundice at operation; and 4 (1.9 per cent.) had jaundice at operation, but there is no note as to whether or not there were previous attacks. 71 (32.8 per cent.) had jaundice at operation; 141 (65.3 per cent.) had no jaundice at operation; 121 (56.0 per cent.) had attacks of jaundice prior to the time of operation. 116 (53.7 per cent.) had jaundice and gallstones; 21 (10.0 per cent.) had jaundice, but no gallstones; 60 (27.7 per cent.) had gallstones, but no jaundice; 11 (5.0 per cent.) had no jaundice and no gallstones; and in 8 (3.6 per cent.) there is no statement as to the association of jaundice and gallstones. 12 (5.5 per cent.) had jaundice and colicky pains, but no gallstones.

Of 60 patients who had gallstones but no jaundice, 44 (73.3 per cent.) had colicky pains; 12 (20.0 per cent.) had non-colicky pains; and in 4 (6.7 per cent.) the character of the pain is not accurately described.

Of 11 patients who had no jaundice and no gallstones, 8 (72.7 per cent.) had non-colicky pains, and 3 (27.3 per cent.) had colicky pains.

Of 34 patients that had no gallstones, 12 (35.3 per cent.) had no jaundice at operation, but did have previous attacks; 11 (32.4 per cent.) never had jaundice; 9 (26.5 per cent.) had jaundice at operation and previous attacks; 1 (2.9 per cent.) had jaundice at operation, but no previous attacks; and in 1 (2.9 per cent.) there is no statement as to the occurrence of jaundice.

In a general way we may say that jaundice may be due to one of two factors: (1) Mechanical obstruction of the biliary passages, and (2) some disturbance in the bile-secreting function of the liver. The mechanical obstructions comprise (1) those due to a gallstone; (2) those due to inflammatory swelling of the lining of the biliary ducts, and (3) those due to compression of the common duct or of the extrahepatic part of the hepatic duct by a large stone in the cystic duct, swollen lymph glands, regional tumors, inflammatory exudations, adhesions, kinking of the ducts, etc. We may, therefore,

speak of a lithogenous jaundice, an inflammatory (or infectious jaundice) and a compression jaundice—ill-advised as these terms may be on some occasions. The practical deductions to be drawn from the known facts are (1) that in some cases of cholelithiasis jaundice is not due to obstruction of the ducts by a stone, but rather to infection, inflammatory swelling of the ducts, and (2) that in many cases of cholelithiasis jaundice does not occur at all, although other important and distressing symptoms may be quite obtrusive. Of the 216 patients under review, 74 (34.2 per cent.) never had jaundice; 141 (65.3 per cent.) had no jaundice at the time of operation, whereas 121 (56 per cent.) gave a history of attacks of jaundice prior to the time of operation. These, in some cases, antedated the operation many years; in some cases long periods of freedom from jaundice followed; in some cases there were no subsequent attacks of jaundice whatever. Remembering, then, that jaundice may be entirely absent, and that when it occurs, it may be late, inconstant, and inconspicuous, we should train ourselves to recognize cholecystitis and cholelithiasis in the absence of jaundice—and of gallstone colic.

The Size of the Gall-bladder. Of 216 patients—in 88 (40.7 per cent.) the gall-bladder was enlarged; in 9 (4.1 per cent.) the gall-bladder was normal in size; in 32 (15.0 per cent.) the gall-bladder was small and atrophic; and in 87 (40.2 per cent.) the size of the gall-bladder is not mentioned in the case-history.

Of 88 patients in whom the gall-bladder was enlarged, 74 (84.0 per cent.) had gallstones, and 14 (16.0 per cent.) had no gallstones. 49 (55.6 per cent.) had jaundice, 36 (41.0 per cent.) had no jaundice, and in 3 (3.4 per cent.) the presence or absence of jaundice is not mentioned.

Of 9 patients in whom the gall-bladder was normal in size, 7 (77.7 per cent.) had gallstones, and 2 (33.3 per cent.) had no gallstones. 7 (77.7 per cent.) had jaundice, and 2 (33.3 per cent.) had no jaundice.

Of 32 patients in whom the gall-bladder was small and atrophic, 30 (93.7 per cent.) had gallstones, and 2 (6.3 per cent.) had no gallstones, but did have fistulæ. 25 (78.1 per cent.) had jaundice, and 7 (21.9 per cent.) had no jaundice.

Of 87 patients in whom the size of the gall-bladder is not mentioned, 71 (81.6 per cent.) had gallstones, and 16 (18.4 per cent.) had no gallstones. 57 (65.4 per cent.) had jaundice, 29 (33.4 per cent.) had no jaundice, and in 1 (1.1 per cent.) the presence or absence of jaundice is not mentioned.

Of 116 patients that had jaundice and gallstones—in 47 (40.5 per cent.) the gall-bladder was enlarged, in 31 (26.7 per cent.) the gall-bladder was small and atrophic, in 4 (3.5 per cent.) the gall-bladder was normal in size, and in 34 (29.3 per cent.) there is no mention of the size of the gall-bladder.

Of 60 patients that had gallstones, but no jaundice—in 29 (48.3 per cent.) the gall-bladder was enlarged, in 10 (16.7 per cent.) the gall-bladder was small and atrophic, in 2 (3.3 per cent.) the gall-bladder was normal in size, and in 19 (31.7 per cent.) there is no mention of the size of the gall-bladder.

Of 21 patients that had jaundice but no gallstones—in 8 (38.1 per cent.) the gall-bladder was enlarged, in 3 (14.3 per cent.) the gall-bladder was small and atrophic, in 2 (9.5 per cent.) the gall-bladder was normal in size, and in 8 (38.1 per cent.) there is no mention of the size of the gall-bladder.

Of 11 patients that had neither jaundice nor gallstones—in 7 (63.3 per cent.) the gall-bladder was enlarged, in 3 (27.3 per cent.) the gall-bladder was small and atrophic, and in 1 (9.0 per cent.) the gall-bladder was normal in size.

Of 47 patients that had jaundice, gallstones, and enlargement of the gall-bladder—in 26 gallstones were present in the gall-bladder alone; in 5, gallstones were present in the gall-bladder and cystic duct; in 5, gallstones were present in the gall-bladder and common duct; in 4, gallstones were present in the cystic duct alone; in 4, gallstones were present in the common duct alone; in 2, gallstones were present in the gall-bladder, hepatic and common ducts; in 1, gallstones were present in the gall-bladder, cystic, hepatic, and common ducts.

Of 31 patients that had jaundice, gallstones, and a small and atrophic gall-bladder—in 10, gallstones were present in the gall-bladder alone; in 7, gallstones were present in the common duct alone; in 8, gallstones were present in the gall-bladder and common duct; in 2, gallstones were present in the gall-bladder and cystic duct; in 2, gallstones were present in the cystic duct alone; in 1, gallstones were present in the adhesions (not otherwise specified); in 1, gallstones were present in the gall-bladder, hepatic and common ducts.

Of 4 patients that had jaundice, gallstones, and a gall-bladder normal in size—in 3, gallstones were present in the gall-bladder alone, and in 1, gallstones were present in the gall-bladder and common duct.

Of 29 patients that had gallstones and enlargement of the gall-bladder, but no jaundice—in 24, gallstones were present in the gall-bladder alone; in 3, gallstones were present in the gall-bladder and cystic duct; and in 2, gallstones were present in the cystic duct alone.

Of 10 patients that had gallstones and a small and atrophic gall-bladder, but no jaundice—in 9, gallstones were present in the gall-bladder alone, and in 1, gallstones were present in the adhesions (not otherwise specified).

Of 2 patients that had gallstones and a gall-bladder normal in size, but no jaundice—in 1, gallstones were present in the gall-bladder alone, and in 1, gallstones were present in the gall-bladder and cystic duct.

Of 34 patients that had no gallstones—in 14, the gall-bladder

was enlarged; in 2, the gall-bladder was normal in size; in 2, the gall-bladder was small and atrophic, and in 16, there is no mention of the size of the gall-bladder.

The clinical significance of the size of the gall-bladder has engaged the attention of physicians and surgeons for a long time, but special consideration has been given to its diagnostic importance since a knowledge of what is known as Courvoisier's law has become general. As is well known, Courvoisier, basing his opinion upon an analysis of 187 cases, stated that in cases of chronic jaundice contraction of the gall-bladder is suggestive of gallstones, and that dilatation of the gall-bladder is suggestive of biliary obstruction caused by factors other than gallstones—that is, pressure from without, most often carcinoma of the head of the pancreas. This so-called law is generally made use of nowadays in diagnosis, and serves us well in the majority of cases, although as Courvoisier himself admitted, there are some notable exceptions to its universal applicability. Unfortunately, the case-histories under review are not in all respects sufficiently detailed to permit a final statement in this particular; but it is of some interest to know that in at least 40.5 per cent. of 116 patients that had gallstones and jaundice the gall-bladder was enlarged. In somewhat more than one-half of these cases, however, the gallstones were present in the gall-bladder alone, and the associated jaundice was doubtless due to inflammatory swelling, pressure from without, etc. I would not, therefore, call into question the general trustworthiness of Courvoisier's law—of the value of which there is ample corroborative testimony—but I should suggest the value of continued study of the conditions found at operation, and emphasize the importance of basing our diagnoses upon the varying grouping of the symptoms in the different cases, rather than upon any single symptom or sign. I have studied a sufficient number of gall-bladders to convince me that Courvoisier's explanation of the shrinkage of the gall-bladder in these cases is correct: that is, that it is due to inflammatory thickening and cicatrization the consequence of repeated infection. This process requires time—whence the condition of the gall-bladder varies early and late in the disease; and that such cicatrization does occur is suggested by its presence in a number of our cases in which gallstones were present without jaundice. It is manifestly impossible that such a thickened and cicatrized gall-bladder should dilate no matter what the obstruction in the common duct. One can conceive, however, of an acute cholecystitis or empyema of the gall-bladder (with distention) and an acute impaction of a stone in the common duct (with jaundice); or of hydrops or empyema of the gall-bladder (with distention) and a large stone in the cystic duct compressing the common duct (with jaundice); or of the association of a stone in the common duct and carcinoma of the head of the pancreas—in which event the gall-bladder might be dilated, or

small, thickened, and atrophic or sclerosed. The practical inference is that, although as maintained by Courvoisier his law may hold good in 80 per cent. of cases, a patient with chronic jaundice and enlargement of the gall-bladder should not be uncerimoniously condemned to the diagnosis of carcinoma of the pancreas, but that the grouping of the other symptoms should determine the diagnosis and suggest the treatment.

THE FEVER. Of 216 patients, 143 (66.2 per cent.) had fever some time during the course of the disease; 65 (30.1 per cent.) had no fever; and in 8 (3.7 per cent.) there is no note of the presence or absence of fever.

Of 143 patients who had fever, 73 (51.0 per cent.) had gallstones and jaundice; 45 (31.5 per cent.) had gallstones, but no jaundice; 16 (11.2 per cent.) had jaundice, but no gallstones; and 9 (6.3 per cent.) had no gallstones and no jaundice.

Of 65 patients who had no fever, 42 (64.6 per cent.) had gallstones and jaundice; 14 (21.5 per cent.) had gallstones, but no jaundice; 7 (10.8 per cent.) had jaundice, but no gallstones; and 2 (3.1 per cent.) had no gallstones and no jaundice.

As regards the fever, I desire only to point that it should be correctly interpreted; that it is a manifestation of infection and is not due merely to reflex causes, nervous perturbations, etc., as was at one time thought. In many cases, concurrently with the development of the so-called gallstone colic, there is a sudden elevation of the temperature, followed sometimes by an equally sudden fall; in these cases, the onset of the fever may or may not be associated with chilliness or a definite chill. This as well as the more continuous fever of frank acute cholecystitis and the recurring paroxysmal fever, known as Charcot's biliary intermittent fever, a characteristic feature of chronic calculous disease (cholecystitis, cholangitis, cholelithiasis), are infectious in nature; whence it follows that in the clinical study of our patients we should interpret the occurrence of fever as clear evidence of infection.

NON-CALCULOUS CHOLECYSTITIS. Of the 216 patients under review, 34 (15.8 per cent.) had no gallstones.

Of 34 patients who had no gallstones, 12 (35.3 per cent.) had no jaundice at operation, but did have previous attacks; 11 (32.4 per cent.) never had jaundice; 9 (26.5 per cent.) had jaundice at operation and previous attacks; 1 (2.9 per cent.) had jaundice at operation, but no previous attack; and in 1 (2.9 per cent.) there is no statement as to jaundice. 24 (70.6 per cent.) had colicky pains; 10 (29.4 per cent.) had non-colicky pains. 21 (62.0 per cent.) had fever; 13 (38.0 per cent.) had no fever. 14 (41.2 per cent.) had a distended gall-bladder; 2 (5.9 per cent.) had a normal-sized gall-bladder; 2 (5.9 per cent.) had a small and atrophic gall-bladder, and in 16 (47.0 per cent.) there is no statement as to the size of the gall-bladder. 19 (56.0 per cent.) had adhesions; 5 (14.7 per

cent.) had no adhesions, and in 10 (29.3 per cent.) there is no statement as to the presence or absence of adhesions. 12 (35.3 per cent.) had colicky pains and adhesions; 6 (17.6 per cent.) had adhesions and non-colicky pains; 4 (11.8 per cent.) had colicky pains and no adhesions; 1 (2.9 per cent.) had non-colicky pains and no adhesions, and in 11 (32.4 per cent.) there is no note of the association of pains and adhesions.

ADHESIONS OF THE UPPER ABDOMEN.

I must now ask your attention to adhesions of the upper abdomen—in the neighborhood of the gall-bladder and the biliary ducts. These adhesions have frequently been encountered by pathologists and surgeons, but it is only recently that we are awakening to a knowledge of their full import. I have little hesitancy in confessing that in years gone by I frequently observed and commented upon the presence of such adhesions, but so little did I apprehend that they possessed noteworthy clinical significance, that I scarcely make a note of them in my necropsy protocols; recognizing their presence I was not alive to their significance. Their production is intimately associated with inflammatory phenomena of the organs of the upper abdomen—the gall-bladder, the biliary ducts, the stomach, the duodenum, etc.; but of all organs, the biliary tract is doubtless the chief offender. In most cases, these adhesions represent the sequels of past subacute and chronic, sometimes acute, inflammation of the organs hereabout, and they are thus a direct result, an expression, of infection.

The conditions are quite analogous to those in and about the vermiform appendix. Should the biliary infection be acute and virulent, the bacteria or their toxins penetrate the wall of the gall-bladder or the biliary ducts and engender a fibrinous exudation, a more or less local peritonitis. In the event of the later development of suggestive clinical symptoms, much diagnostic significance attaches to a history of such past infections (typhoid fever, definite cholecystitis, etc.).

In the subacute and chronic infections, however, the formation of adhesions goes on less obtrusively, often entirely latently—whence the symptoms that develop later are commonly misinterpreted. In consequence of milder infections the less virulent toxins give rise to less desquamation of peritoneal endothelium, to the exudation of less fibrinous lymph, and to less inflammatory phenomena —sufficient, however, often to lead to more or less widespread adhesions. Frequently, these are quite localized and very delicate (gall-spiders, resembling spider webs, they have been aptly called by Morris[1]); in other cases they are quite widespread and very dense.

[1] Bile Tract Adhesions, Med. Record, 1905, lxvii, 447.

All gradations are encountered—from those very delicate and so slight as scarcely to occasion comment or awaken interest and possibly, though certainly not always, innocuous clinically, to others so dense and unyielding that the wonder is that the patient has been able so long to withstand their effects. Of the 216 patients under review, 123 had adhesions, 22 had no adhesions, and in 71 the presence or absence of adhesions is not mentioned (probably none were present).

The clinical recognition of these adhesions is sometimes a matter of difficulty, but is perhaps not impossible in the majority of cases. It is essential, in the first place, that we recognize that many of the cases of so-called "stomach ache," "biliousness," indigestion, gastralgia, gastritis, gastroduodenitis, persistent or recurring vomiting, constipation, etc., in reality have an anatomical basis. We now know that many of these cases are due to an unsuspected or latent gastric or duodenal ulcer, definite cholecystitis, chronic pancreatitis, etc.; in other cases, they are due to pericholecystic, perigastric, periduodenal, and pericolonic adhesions. We must study attentively patients who complain of general ill-health, with more or less ill-defined gastric or epigastric symptoms—epigastric distress and tenderness, sometimes associated with nausea and vomiting, and perhaps constipation. Attacks of such symptoms may come on periodically without definite cause; they may last a few days, and cease quite as causelessly. Now and then the attack may be ushered in with chilliness (rarely a definite chill), and may be attended by a little fever. Examination may reveal some tenderness in the epigastrium, perhaps a little to the right of the median line, toward the region of the pylorus or the gall-bladder, and some rigidity of the overlying muscle. Deep pressure is sometimes very painful. Later, should the pylorus or the duodenum become obstructed, the commoner manifestations of dilatation of the stomach supervene; but these need not detain us now, since I am more interested in the latent, masked, and unsuspected cases. Especial diagnostic importance attaches to intractability of the symptoms and a history of past infection of the biliary tract (typhoid fever, cholecystitis, cholangitis, jaundice, etc.). In the event of suspecting such adhesions, we must exclude other factors that may cause similar symptoms, remembering, however, that in many cases symptoms supposedly due to disease of the stomach or intestine are due disease of the gall-bladder or adhesions of the upper abdomen.

THE GENERAL PRINCIPLES OF TREATMENT.

The intelligent treatment of any patient presupposes a clear conception of (1) the pathology of the disease in question, of its pathological anatomy, and its natural tendencies; and (2) of the

means of treatment at our command and the objects attainable by their use. Of these none is more important than a knowledge of the natural history of the disease. To attempt by internal medication to dissolve a gallstone that is insoluble; to cause the passage through the gall ducts of a gallstone when the ducts are impassable to a stone of its size; to attempt to cure supposed gastric symptoms by measures directed to the stomach when the cause of the symptoms is adhesions about the gall-bladder, and to cause the solution of these adhesions when, perchance, they have been diagnosticated—is as futile as it is irrational.

In view of the frequency of gallstones in infections of the biliary tract they may well be included in a consideration of the indications for treatment. The main indications may be said to be: (1) To cause solution of the stone or stones; (2) to cause the discharge of the stone or stones; and (3) to treat the complicating infection.

Solution of the Gallstones. As regards the solubility of the gallstones Naunyn[1] states that we have to consider cholesterin, bilirubin-calcium, calcium carbonate, and calcium phosphate. Of these, bilirubin-calcium and the inorganic salts of calcium are insoluble in the bile; cholesterin, however, is quite soluble in the bile, and even in conditions of disease the bile scarcely if ever contains so much cholesterin that it cannot take more in solution. We have, therefore, to admit the possibility of cholesterin stones being dissolved. In fact, there is some experimental evidence that goes to prove that not only *in vitro*, but also *in vivo*, cholesterin stones may be dissolved. Naunyn quotes Labes' experiments of thirty years ago, in which he introduced gallstones into the gall-bladder of dogs and, killing the animals two months later, found that partial solution of the stones had occurred. Recently, Bain[2] has published the results of some studies that show the same possibility. In these experiments, however, the calculi were introduced into normal gall-bladders (or, as in one or two of Bain's experiments, into gall-bladders in which a cholecystitis has been artificially produced). The conditions are quite different in the human subject affected with gallstones—in which we have to deal not only with the stones, as such, but also with the subacute or chronic lithogenic catarrh—the direct cause of the gallstone formation. It is thus exceedingly doubtful whether spontaneous solution of gallstones ever occurs in the human subject—indeed, whether it is at all possible. Naunyn states that in studying one thousand cases of cholelithiasis he has seen trustworthy evidence of the solution of the stone in perhaps ten cases (1 per cent.). Though he does

[1] Zur Naturgeschichte der Gallensteine und zur Cholelithiasis, Mith. Grenzgeb. Med. u. Chir., 1905, xiv, 537.

[2] An Experimental Contribution to the Treatment of Cholelithiasis, Brit. Med. Jour., 1905, ii, 269.

not deny that the solvent action of the bile may be a contributing factor in the process, he inclines much more to the belief that the disintegration of the stone is due to the action of bacteria, and for the reasons—that he has found this disintegration, not in cases of a single stone, but in cases of numerous stones; that the process involved many stones; that the solution was not circumferential, as it would be in the event of solution in a dissolving menstruum, but was circumscribed at the periphery and extended deep into the calculi; and that the disintegration went hand-in-hand with the deposition in the stones of inorganic calcium (phosphates and carbonates), certainly due to bacterial activity. The reasons for the non-occurrence of solution of the stones are, (1) and most important, the fact that on account of the presence of the stones the activity of the walls of the gall-bladder is compromised, it cannot empty itself completely (a certain amount of residual bile is always present), and the desirable irrigation of the gall-bladder with fresh bile (the solvent menstruum) does not occur; and (2) the fact that on account of the associated catarrh of the biliary tract the bile contains more cholesterin than is normal, and thus takes up an additional increased amount with less avidity.

Naunyn furthermore points out that even should it be possible to cause disintegration of the stones in the gall-bladder, it is exceedingly doubtful how much good would be thereby accomplished—since in this event the residual particles of the disintegrated calculi would remain in the gall-bladder, and form the nucleus of new calculi. These are formed by the cementing together or incrustation of the disintegrated particles by newly precipitated bilirubin-calcium, the formation of which is favored by the persisting infectious inflammation of the mucous membrane which gives rise to a secretion rich in calcium.

This reasoning is well borne out by clinical experience, since it is exceedingly doubtful whether, with any means at our command, we can cause solution of gallstones in the gall-bladder. We now know that the thousands of gallstones said to have been passed by the bowel after the administration of olive oil are merely masses resembling gallstones in outward appearances and due to the basely deceptive powers of the olive oil acquired in its passage through the intestinal tract. We must conclude, then, that the solution of gallstones is scarcely the object of rational treatment.

Discharge of the Gallstones. Gallstones may be discharged from the biliary tract *per via naturalis*, or by way of fistulæ. Many stones rarely pass *per via naturalis*, though single stones sometimes do; large stones, if discharged, practically always leave the gall-bladder by devious tracts and the formation of fistulæ. This is part of the natural history of the disease. In some cases the gallstones have been entirely latent until they appear in the stools, in which event if the stone is large, we must assume the formation

of a gall-bladder-colonic fistula, since large stones having ulcerated into the small intestine practically always cause noteworthy symptoms in their downward passage, and not infrequently even fatal intestinal obstruction. In the event of a single stone only being present, the possibility of cure is thus presented, but the possibility is too remote for serious consideration. It is an occurrence which should not be hoped for, nor awaited, nor sought by medical means, even were such at our command; it also then cannot be considered an object of rational treatment.

Treatment of the Biliary Infection. There remains as an indication for treatment—the biliary infection. It has been well emphasized by many writers that the one worthy object of internal medication in cholelithiasis is to restore the latency from which the gallstones have been awakened, in other words, to control the infection at the basis of gallstone activity. This must also be the object of medicinal or internal treatment in the absence of gallstones. The infection of the biliary tract is what the physician has to treat. If I have pointed out that he cannot expect to dissolve the calculi nor to cause their discharge *per via naturalis* or by way of fistula-formation, I do not mean to suggest that the physician is hopeless in the presence of calculous and other disorders of the biliary tract. Unquestionably he can do a very great deal of good; in many cases lead his patient into a condition of virtual cure, that is, latency, or sterility of the biliary tract; but he must also recognize his limitations, and by a judicious balancing of his abilities and limitations not subject his patient too long to a useless medicinal treatment when early surgical intervention may restore him to health, whereas delayed it may not only add to the miseries of a miserable existence, but actually hasten the final termination.

Time forbids that I discuss in detail the medicinal treatment of infections of the biliary tract, or that I point out the manner whereby the use of Carlsbad (and other similar) waters, salicylic acid preparations, the iodides, podophyllum, and other cholagogues, certain antispasmodics, etc., do good in many cases; but I may state that the favorable result of medicinal treatment in the vast majority of cases by no means indicates the cure of the disease, but merely the restoration of latency, and this, of course, is by no means an undesirable achievement. By promoting the free flow of bile we may effect the discharge of infecting bacteria and their toxins from the biliary passages and prevent their ascent to the smaller ducts, and by the constant irrigation thus produced, as well as by other measures, we may allay the inflammatory phenomena. Ultimately, the biliary tract may be rendered sterile, and should the inflammatory phenomena subside completely a condition of latency, quiescence of the gallstones, results. In rare cases this latency is permanent, and the patient experiences no further complaints. In most cases a low-grade chronic catarrh of the gall-

bladder persists; in other cases the gall-bladder being a *locus minoris resistentiæ* readily again becomes infected, and the patient in consequence is continuously or intermittently ill. Should gallstones be passed by the bowel, we must remember that all the gallstones harbored by the patient are seldom, if ever, thus passed, and that even were this the case the persisting catarrh of the gall-bladder is quite certain to give rise to the formation of new stones. A knowledge of the natural history of calculous disease, thus, demonstrates to us unequivocally the objects and the limitations of rational medicinal treatment.

INDICATIONS FOR SURGICAL INTERVENTION.

Apropos of surgical treatment, I may say that by means of a well-timed and properly executed operation, we may remove the gallstones and control the concomitant catarrh; that is, having removed the stones, the infectious catarrh, the cause of the stones, may be controlled and finally cured, and the formation of new stones prevented.

The important question to decide is, When should an operation be undertaken? I am by no means prepared to say that the diagnosis brings with it the indication for surgical intervention. The multiplicity of the pathological lesions and of the clinical manifestations, and their course uninfluenced by operation; the general health of the gallstone subject; the hazard of an operation *per se;* and the personal inclinations of the patient often give us pause.

In many cases the question of early operation scarcely admits of discussion. These are cases in which there is acute cholecystitis (with or without cholangitis—jaundice), with evidences of severe infection, and in which the symptoms and local signs, instead of abating, become more pronounced: these comprise cases of acute cholecystitis developing during or after an attack of typhoid fever or other infectious disease, as well as during the course of previously latent or manifest cholelithiasis, cases in which the diagnosis of severe suppurative or gangrenous cholecystitis with perforation of the gall-bladder or severe local or generalized peritonitis, seems warranted, and in which impending if not actual infection of the hepatic ducts is not unlikely. The risk attending the progress of the disease in such cases is more than the hazard of the operation; indeed, without operation many of these cases would unquestionably soon terminate fatally. Happily, however, most of the acute infections of the biliary tract subside spontaneously or under the beneficent influence of wisely directed medicinal treatment, even many of those that develop very suddenly and in which the outlook during the first twelve or twenty-four hours seems fraught with danger. In general, it is much wiser that the acute manifestations

should subside without operation—which, should it be deemed desirable later, with a view to relieve the basic condition, may then be undertaken with much more reasonable hope of ultimate success.

Immediate operation is imperative also in the event of acute intestinal obstruction in a cholelithitic subject, or in a person in whom adhesions have been suspected.

Operation, though not necessarily always immediate, is indicated in all cases of persistently enlarged gall-bladder—whether the enlargement is due to simple hydrops (serous and mucous fluid), empyema (pus), or gallstones.

The question of when to operate in cases of gallstones that have become or have been active must be decided in each individual case; but one should bear in mind that, as a rule, it is not the many stones in the gall-bladder that do the most damage, but the one or two stones that get into the ducts, that the special risk attending operations for gallstones increases with delay, and that whereas gallstones are readily removed from the gall-bladder, their removal from the ducts is commonly attended by serious difficulties and is sometimes impossible. The time of election for operation, therefore, is before the stones have entered the ducts. This is by no means always possible, nor indeed desirable. Thus, I should not advise the removal of stones that are and always have been quiescent. If, however, the gall-bladder is enlarged, and the stones have given warnings of discontent with their hampered quarters—local discomfort and distress—operation should be undertaken. Nor would I insist upon operation after a single, short, successful gallstone colic (so-called), but I should by no means be averse to it. I should bear in mind that some patients have only a single gallstone colic, but that the majority have many attacks. Personally, however, I am inclined to believe that one severe attack would be sufficient for me, and that having recovered from it I should seek the services of a skilful surgeon. Two, certainly three attacks, I believe, should be looked upon as a positive indication for operative intervention. In the event of hesitation on the part of the patient the pathological changes and the likely course of the disease should be explained to him in simple language, and he should be required to make the decision for or against operation and to assume the responsibility.

In impaction of a gallstone with chronic jaundice operation is called for, and my conviction is for early rather than delayed intervention. As I have said, the risk of gallstone operations is the risk of delay, since with delay serious infections of the biliary tract supervene (with the characteristic intermittent fever, etc.), the tendency to pronounced hemorrhage increases with the persistence of the jaundice, the general nutrition of the patient fails, he becomes less able to withstand the vicissitudes of the operation, and stagnation of the bile leads to dilatation of the ducts and seriously

compromises the functional activity of the liver cells. The decision as to the time of operation must be made for each individual patient, but I can scarcely conceive of any advantage commonly attending a delay beyond one or two weeks, and this, too, in full knowledge of the fact that some patients have recovered without operation after a much longer period.

Finally, operation is indicated in those obscure cases usually diagnosticated indigestion, biliousness, gastralgia, recurring vomiting, etc., in which with reasonable certainty the presence of pericystic, perigastric, or periduodenal adhesions may be surmised.

In each of the foregoing classes of cases, operative measures attain that which is impossible of attainment, or attainable only with difficulty, slowly, and imperfectly, with medicinal measures—whence surgical measures find their justification. They find additional justification in the fact that early intervention tends to prevent the development of certain complications and sequels, such as extensive adhesions, fistulæ, carcinoma of the gall-bladder, acute and chronic pancreatitis, etc. An operation, however, should not be lightly undertaken in persons suffering with divers disorders of nutrition, such as gout, obesity, so-called chronic rheumatism, diabetes, etc.; nor in those with faulty kidneys, lungs, heart, etc.; the risk attending the anesthesia and the shock of the operation is too great and the recuperative power of the patient too much reduced, to expect from operation a great deal more than can probably be attained by well-directed medicinal, hygienic, and dietetic treatment, aside from the fact that in the one instance the patient will remain alive though indisposed, while in the other he may be much longer dead. Finally, the fact that the operative results are not always what were hoped for, is not in itself a contra-indication to operation; on the contrary, these untoward results are often attributable rather to the fact that in many long-delayed cases the anatomical lesions are such as to be almost, if not quite, irremediable by any and all means at our command.

REVIEWS.

CLINICAL OBSTETRICS. By ROBERT JARDINE, Professor of Midwifery in St. Mungo's College. Glasgow and London: Rebman, Ltd., 1905.

THE work is essentially a clinical treatise and is peculiar for the narration to a considerable extent of interesting cases. The first section of the book is occupied with the complications of pregnancy; then follows a brief description of labor and its mechanism with the management of parturition and the puerperal period. The author then treats of the hemorrhages of pregnancy, of complications in labor and the puerperal period, obstetric operations, accidents and diseases of the newborn child, the feeding of the infant, and concludes with the statistics of thirty years' work in the Glasgow Maternity Hospital.

In treating of the pernicious nausea of pregnancy the author doubts the influence of uterine displacement in producing this condition. He accepts the theory of toxemia as most satisfactory in the present stage of our knowledge. The use of saline solutions is especially recommended. We have found in these cases especial value in the examination of the blood, as affording an index of the gravity of the patient's condition. When extensive disintegration of blood corpuscles is present with the clinical phenomena of pernicious anemia, pregnancy must be terminated at once. The author, however, seems to have relied entirely upon the clinical phenomena presented.

In dealing with nephritis during pregnancy the author has found benefit in adding to the ordinary salt solution the acetate of sodium in equal proportions with the chloride. In describing cases of pernicious anemia in pregnancy he draws attention to enlargement of the liver with hemorrhages into the parenchyma, and purpura, these indicating that toxemia of hepatic origin is the most important cause of the anemias of pregnancy.

In dealing with cardiac complications during pregnancy strophanthus in the author's cases gave especially good results as a cardiac tonic. The interruption of pregnancy is frequently demanded, and the author would accomplish this before midterm, as the heart is usually then best able to bear the strain of labor. In dealing with phthisis complicating pregnancy the author does not insist upon the termination of pregnancy in all cases. With the success

of modern methods in the treatment of tuberculosis and the inevitable progress of the disease in the mother during gestation we believe that pregnancy in tuberculous patients should be interrupted as soon as possible, whereupon the mother's chance for recovery will be greatly improved.

In conducting the second stage of labor the illustration represents the physician's hand as completely covering the perineum. In our experience this predisposes to laceration, and we believe it to be important that an inch of the perineum be left without support, the hand being placed across the pelvic floor, the anus covered with antiseptic gauze or cotton. The author would immediately close all lacerations of the perineum and pelvic floor, including the cervix if it be badly torn. He states that the cervix can be drawn down and stitches inserted easily. This coincides with our experience, and we have seen nothing but good results from the immediate closure of lacerations.

In describing the hemorrhages of the later months of pregnancy the author reports a case of induced labor in which a de Ribes' bag burst under pressure and caused separation of the placenta and severe hemorrhage. The case proved fatal, but an autopsy could not be obtained. In concealed internal hemorrhage he has found the results of the ordinary rapid delivery exceedingly bad. He believes that delivery by abdominal section promises better. In treating of rupture of the uterus, he says he has seen this accident occur in cases in which the physician failed to deliver with forceps and has then resorted to turning. In the treatment of postpartum hemorrhage he has found suprarenal extract of advantage and does not recommend the use of iron. He has also been successful in tamponing firmly the uterus and vagina. He describes and illustrates a simple apparatus for giving saline infusions. In dealing with traumatic hemorrhage after labor, the author states that some of the worst cases of hemorrhage which he has seen have followed spontaneous delivery, or version and extraction, before the cervix was fully dilated. He would immediately close the cervix after hemorrhage and also for considerable or extensive laceration without hemorrhage. In dealing with rigidity of the cervix complicating labor the author depends upon manual dilatation under chloroform or hot vaginal douches. He does not mention the use of morphine, which is very successful in many of these cases.

In treating of contracted pelves he draws especial attention to the value of palpating the pelvis with the hand. He considers the most accurate method of measuring the size of the conjugate is with the fingers or with the closed hand introduced within the pelvis. He also draws attention to the value of Walcher's position in increasing the size of the pelvis. He recognizes the value of the Cesarean operation in dealing with contracted pelves, but in cases in which version and forceps are permissible he would use version in posterior parietal

presentations, reserving the use of forceps for anterior parietal presentations. He draws attention to injury to the child occurring from delivery in contracted pelvis through pressure upon the cranium. In selecting operation in disproportion between the head and the forceps he would try forceps in Walcher's position. Failing this, with the child in good condition, he would employ symphyseotomy. In subsequent labors pregnancy might be interrupted.

The author believes the cause of eclampsia to be a chemical substance the product of tissue metabolism. He still adheres to the nephritic origin of eclampsia, and in one case in which convulsions and labor occurred without albumin in the urine, he does not believe that true eclampsia was present. He has, however, noted the fact that a patient may have convulsions without albumin in the urine or with a very small quantity only. He does not accept the modern hepatic pathology of this condition. In treatment, with the use of salt solution he has employed bicarbonate of potassium with chloride of sodium by hypodermoclysis, but now uses acetate of sodium and chloride of sodium in equal parts. He has found great advantage from bleeding followed by transfusion, and believes that the less interference with the uterus in eclampsia the better for the patient. In this we agree with him. He does not recognize the hemorrhagic conditions, notably in the lung, following eclampsia, which are the usual causes of the fatal issue.

In the treatment of puerperal septic infection after emptying the intestine the author advises the use of quinine and Dover's powder for pain. He considers strychnine given hypodermically the best cardiac stimulant. He does not favor curetting, but would douche the uterus with 1 to 2000 bichloride, followed by boiled water. If necessary, he would repeat intrauterine douches daily; he has not seen mercuria poisoning from this treatment. We cannot join him in his recommendation of repeated intrauterine douches nor in the use of bichloride of mercury within the womb. In pyemia he has found good results from tamponing the uterus with iodoform gauze. In sapremia after irrigating the uterus he applies iodine freely to the interior. He urges the importance of the upright or semi-upright posture in securing drainage in these cases, and describes the case of a patient suffering from severe sapremia, who persisted in leaving her bed and sitting up. The patient was better as soon as she assumed the sitting posture. He has used antistreptococcic serum, but is not sure of its value. He reports fully a number of interesting cases of puerperal sepsis, including tetanus, in which pre-existing foci were present.

The author has performed symphyseotomy eight times with success. One of his cases was complicated, a laceration of the vagina from a fractured end of the pubes, and another patient had separation of the urethra from the vagina. This was treated by inserting a catheter, by bringing the urethra down and stitching it in its

normal position. In choosing the time for Cesarean section the author would operate as soon as the cervix is obliterated and the os will admit one finger. He does not rupture the membranes before operation. For controlling hemorrhage, he recommends a large pessary which, being pressed down upon the uterus, controls the circulation efficiently. It should be removed as soon as the uterus has been opened. His uterine stitches include the muscle and peritoneum, but he does not insert separate sutures for the peritoneum. He avoids contact with the interior of the uterus and trusts to a firm binder to keep the dressing in position. He does not use salt solution in the abdomen during the operation. Porro's operation is sometimes selected because it can be done quickly. Cœliohysterectomy is also preferred when further pregnancy is not to occur.

The author contributes two full and interesting chapters upon injuries and malformations of the newborn child. The volume concludes with a chapter upon infant feeding. Appended are the statistics of the Glasgow Maternity Hospital for three decades, a table of 41,111 cases. Among these, 137 deaths occurred, due primarily to parturition, of which 46 resulted from septicemia. At present rigid antiseptic precautions are adopted during labor and the puerperal period. Examinations are made as rarely as possible. The result has been a gratifying decrease in the number of infections.

This book should prove especially valuable to experienced men engaged in obstetric practice. Its cases are fully and clearly described and important clinical points are thoroughly brought out. It will be of less value to the student of medicine until he becomes a resident in a hospital. The style is clear and interesting, and it will prove a welcome addition to obstetric literature. Its popularity is already attested by the appearance of a second edition.

E. P. D.

A Text-book of Physiology. By William H. Howell, Ph.D., M.D., LL.D., Professor of Physiology in the Johns Hopkins University, Baltimore. Philadelphia and London: W. B. Saunders & Co., 1905.

At the present time it is almost impossible in any department of science to embody in a text-book for students all the known facts, as well as the conflicting theories, concerning disputed and unsettled points. To include everything, statements must be so brief and concise that to the beginner they lose their force and meaning. The reduction in material, as the author states in his preface, "should be made by a process of elimination rather than by condensation."

This seems a very wise way out of the difficulty and a most successful one, as the book itself shows. From the author's practical experience and wide knowledge he is enabled to select certain phases of his subject which must be expanded sufficiently to make them perfectly clear, even if less important matter must be omitted. This scheme has been carried out with an excellent result. Many antiquated theories, many long and complicated hypotheses to explain unimportant points, are passed by with little more than respectful mention, while the more real and growing subjects are often discussed with some detail. But this selection and elimination is done with the utmost care, and with full knowledge of the worth of what is kept and the value of what is cast away.

In the selected subjects the material is carefully worked over; it is modern and suggestive in its treatment, so that the reader obtains a clear and definite idea of the subject as it stands today. The sections on the central nervous system, the organs of circulation, the respiration, and digestion deserve special mention.

Throughout the text there are many illustrations which assist greatly in explaining certain points. Excellent use is made of cardiographic tracings and charts of various kinds. A good feature, too, is the references to orginal articles inserted as foot notes on almost every page. The delightfully clear style makes reading easy and adds much to the general character of the work. The book can be very highly recommended; it is really of great value. W. T. L.

A HAND-BOOK OF PHYSIOLOGY. By AUSTIN FLINT, Professor of Physiology in the Cornell University Medical College. New York and London: The Macmillan Company, 1905.

THIS book presents the subject of physiology as taught the students of the Cornell University Medical College. In several features it is very interesting. The historical side of physiology is especially brought into prominence, and the work of Harvey, Malpighi, Haller, Du Bois-Reymond, Bernard, and such pioneers is frequently cited, especially in introducing the various subjects which these men illuminated. The anatomical facts bearing upon physiology are given a relatively large place and the illustration of the histology of the more important organs form a special feature. These illustrations are reproductions of three-color photomicrographs—a process used for the first time in this kind of work—and are collected at the end of the book as an atlas of sixteen plates. Although they are an improvement over ordinary photomicrographs as means of illustrations, they still possess much of the indefiniteness of outline and lack of character which make them rather unsatisfactory.

Gross anatomy and histology are being so well taught now that it is a question if the author has not given this side of his work too much emphasis. The book will probably prove rather ill-suited for the modern medical student, as one is struck by the author's preference for broad statements, rather than for recording briefly scientific facts. In several particulars the book does not reflect the most advanced knowledge of the day. The more recent work on the chemistry of digestion finds no place. Enzymes, or ferments, receive very meager notice, and neither word appears in the index. The author does not take sides with either the myogenic or neurogenic theory of the heart-beat, and the recent work in this part of the science receives no notice.

The style of Dr. Flint is very good and the book reads well. His experience of nearly fifty years as a teacher has enabled him to present the subject in an interesting and lucid manner, and for those who wish to study the more elementary phases of physiology, especially from the historical point of view, the book will be found pleasing and profitable. G. C. R.

A System of Medicine by Many Writers. Edited by Thomas Clifford Allbutt, M.D., F.R.S., Regius Professor of Physic in the University of Cambridge, and Humphry Davy Rolleston, M.D., F.R.C.P., Physician to St. George's Hospital and to the Victoria Hospital for Children, London. New edition, revised throughout with additions. Vol. I. New York and London: The Macmillan Co., 1905.

Owing to the rapid strides with which the science of medicine is at present advancing the life of a modern medical book must needs be short. To ensure continuous utility, frequent revisions are necessary. Hence, it has been deemed advisable to bring out a new edition of Allbutt's *System of Medicine*. It is intended that one volume of the series shall be completed each year, thus gradually replacing the 1901 edition.

Volume I of the new series comprises Prolegomena and Fevers. In many a book the preface is no less interesting than the story itself; and as regards that of the volume under review, which is to a certain extent a preface to the whole series, the foregoing statement holds good. In it we find many subjects of great interest discussed, such as, for obvious reasons, are omitted from text-books of smaller scope. Thus an extremely interesting chapter on Anthropology and Medicine has been contributed by Beddoe, which contains much food for reflection. The chapter on the Laws of Inheritance, by Jonathan Hutchinson, is one which will equally repay perusal. The History of Medicine has been tersely and lucidly set forth by Allbutt

and Payne. Under Medical Statistics, Tatham has considered such subjects "respecting the more important diseases of the community" as are of practical utility to the general practitioner. It is to be regretted, however, that no data are given upon arteriosclerosis and heart disease.

The chapters which have undergone the most change in the process of revision are Medical Geography, by Clemow; The Hygiene of Youth, by Dukes; The Dietetics and Therapeutics of Children, by Eustace Smith. Of especial importance are the chapters on the General Pathology of Nutrition, by F. W. Mott, and the General Pathology of New Growths, by F. W. Andrewes, dealing as they do with subjects upon which much research work is being done. J. K. Mitchell has written a valuable article on Massage, a subject with which the physician is often too little acquainted from a practical point of view. Lack of space forbids the mention of many other excellent features of the work. On the whole, it may be said that Volume I is a vast storehouse of medical knowledge classified, grouped, and condensed by eminent authorities so that it shall be available for use with the least possible effort. We have no hesitancy in cordially endorsing the first volume of the revised system, and feel that its character augurs well for the volumes that are to follow. G. W. N.

AN INTRODUCTION TO THE STUDY OF MATERIA MEDICA AND PHARMACOLOGY. By OLIVER T. OSBORNE, A.M., M.D., Professor of Materia Medica, Therapeutics, and Clinical Medicine in Yale University. Pp. 167. Philadelphia and New York: Lea Brothers & Co.

THIS little book includes the elements of medical pharmacy, prescription writing, medical Latin, toxicology, and methods of local treatment. When one considers the field covered in this duodecimo volume he may expect merely a skeleton; but as he reads and studies the book he finds an amount of information, at once essential and accurate, which only the experienced and successful teacher would be able to present. After a few definitions the author proceeds at once to brief notes on experimental pharmacology and early pharmacological research, and then certain drugs for laboratory demonstration, with suggestions for recording experiments; thus suggesting to students the varieties in drug action. Pharmacy opens with definitions and some brief remarks upon the different pharmacopœias, and presents the different classes of preparations in alphabetical order. Toxicology is presented concisely and understandingly. Prescription writing calls for good form, and the results

secured should be satisfactory if the author's methods be followed, for having gone over the grammar of the prescription, its abbreviations, and weights and measures, the evolution of the prescription comes naturally. For the adaptation of remedies to the patient a classification of agents is necessary, and this is briefly and sensibly presented. The questions which arise in dosage are aptly presented and the student will be markedly enlightened by the chapters devoted to special treatments and simple food preparations. Here as well as throughout the book on every page are found many practical points and suggestions. We heartily recommend the book to the student. R. W. W.

THE CLINICAL STUDY OF BLOOD PRESSURE. By THEODORE C. JANEWAY, M.D., Lecturer on Medical Diagnosis, University and Bellevue Hospital Medical College. New York and London: D. Appleton & Co., 1904.

THE importance of accurate instrumental blood pressure measurements is becoming generally recognized, and observations upon the pulse tension are taking their place as routine procedures, with the data relating to the temperature, the respiration, and the pulse. There can be no doubt that if sphygmomanometry were routinely employed in practice, many a case of renal or arterial disease would be diagnosticated which is otherwise overlooked. Hypertension is very constantly present in quite a large number of these cases, whereas urinary findings may vary greatly. In fact, there seems to be an increasing tendency to lay but little importance upon a trace of albumin or a few narrow hyaline casts in the urine.

The appearance of Janeway's book, which is, so far as we know, the only work in English which covers the same ground, cannot but be of signal benefit in directing the attention of the physician to the facts of sphygmomanometry, as thus far known. The volume is divided in parts dealing with the physiological, the technical, and the clinical phases of the question respectively. The subject matter as gleaned from the literature and from the author's personal experience has been well grouped and concisely handled; and has been enhanced in value by a large number of charts and illustrations. The physiological section contains much valuable information which must be borne in mind in attempting to draw deductions from blood pressure observations. In the fourth chapter the advantages and the shortcomings of various forms of apparatus are discussed. The last part of the book deals with blood pressure as encountered in various internal diseases, nervous diseases, surgical and obstetrical conditions, etc. With a work such as this

available there can no longer be any excuse for any practitioner to remain in ignorance of the importance, the value, the technique, and the limitations of sphygmomanometry. G. W. N.

ON LEPROSY AND FISH-EATING. A STATEMENT OF FACTS AND EXPLANATIONS. By JONATHAN HUTCHINSON, F.R.S., formerly President of the Royal College of Surgeons. London: Archibald Constable & Co., Ltd., 1906.

MR. JONATHAN HUTCHINSON has long been known as an earnest and persistent advocate of the theory that leprosy is due in some, as yet unknown, manner to the consumption of badly cured or decomposed fish; and in this volume of more than four hundred pages he presents the evidence for this theory, which he has gathered from literature and from personal observation in India and Africa. With how much earnestness he pursues this subject may be inferred from the fact that, in 1901–2, at a time of life when most men would have given up serious work, he made a journey to South Africa and India for the purpose of gathering new facts, at first hand, in support of this theory.

Mr. Hutchinson believes that there is a close relationship between tuberculosis and leprosy, and that possibly these two diseases are due to the same bacillus. To quote his own words: "It is suspected that the tubercle bacillus (existing in a latent state in many persons and often hereditary) may in certain individuals become modified by some element which exists in badly cured fish, and may be made to assume the activities which result in the phenomena of leprosy." He does not believe that the malady ever arises from direct contact with the sick, and hence regards segregation as unnecessary and cruel. Besides a diet of decomposed fish which is by far the most potent cause in his opinion, he thinks it may occasionally follow the use of food contaminated by the hands of lepers and, in infants, may arise from leprous mothers' milk.

Hansen's discovery of Bacillus lepræ is spoken of as an unfortunate one which "threw back for a quarter of a century the investigation of leprosy. It put investigators on a wrong scent and it suggested a false conclusion," since it added strength to the doctrine of contagion.

Truly, all is fish that comes to the author's net; and many facts which other investigators regard as refuting the fish theory are claimed by him to afford support to his contentions. The well-known fact that, at the present time at least, leprosy is found chiefly in coastal and insular regions is referred to as affording strong presumptive evidence in favor of the fish origin of the disease; but its

occurrence in regions where fish are obtained with difficulty and, therefore, form an insignificant article of diet, as in Natal, presents no difficulties to the author, since, he repeatedly asserts, it is not the quantity of fish consumed that is important, but the quality, even a small quantity of decayed fish being capable of producing leprosy. Recovery is believed to occur in many cases, and relapses are not common except in those living in extreme poverty, or in those who persist in a fish diet. While many interesting and suggestive facts are presented in this book, which, like all that its distinguished author writes, is well worth reading, the evidence is not convincing. M. B. H.

DISEASES OF THE EYE. By G. E. DE SCHWEINITZ, A.M., M.D., Professor of Ophthalmology in the University of Pennsylvania. Fifth edition. Philadelphia and London: W. B. Saunders Co., 1906.

DR. DE SCHWEINITZ'S well known work, of which seven reprints and revisions have appeared since its publication, comes up in this fifth edition to the high standard it has maintained from the beginning. The additions and amendments called for by the progress of ophthalmology serve to keep it abreast with the requirements of the time. As it now stands, it is quite the equal of any single work whose purpose is similar. It is especially commendable from its sense of proportion and sanity of treatment. The author has not been carried away by any of the fads which have been exploited from time to time for a longer or shorter period in the domain of ophthalmology.

Among the thirty-five subjects which appear for the first time we mention the *x*-ray treatment of trachoma, of which the author is not enthusiastic, though he adds that with improved technique and the ability to measure dosage of the rays, the method should be given the fullest trial. Jequiritol is properly said to be not without danger, and should be restricted to old cases of pannus with cicatrized and degenerated conjunctiva. As regards methyl alcohol amaurosis, the author does not decide whether the affection is one of the retina or of the optic nerve, but he suggests that both may be simultaneously affected. The amblyoscope for orthoptic training is commended. This fifth edition is a worthy exponent of the ophthalmology of the present day, and to it the reader can turn with confidence. T. B. S.

URIC ACID: THE CHEMISTRY, PHYSIOLOGY, AND PATHOLOGY OF URIC ACID, AND THE PHYSIOLOGICALLY IMPORTANT PURIN BODIES, WITH A DISCUSSION OF THE METABOLISM IN GOUT. By FRANCIS H. MCCRUDDEN, M.D. The Fort Hill Press, Samuel Usher, Boston, Mass., 1905.

IT is only necessary for one to glance through the book to realize the enormous amount of work required for its preparation. The entire 300 pages or more are taken up with an exposition of the results of innumerable experiments and observations upon the chemistry, physiology, and pathology of one single substance, uric acid. In attempting this, the author has collected all available data from every possible source, and the treatise is a *resume* of practically all the work that has been done upon this subject, with particular reference to recent publications. Original authorities are always quoted and a quarter, often a half, of every page is filled with references. It is, of course, impossible to review the work in detail. The pure chemistry of uric acid and the purin bodies is considered first; next, the physiology is discussed, beginning with nitrogenous metabolism in birds; and finally, the pathological aspect is reviewed, with special reference to gout. The book is primarly one of reference for the advanced student, where he may find the facts relating to almost any part of the subject, with full references to the literature. Though the results of experiment and observation are systematically presented, no attempt is made to draw conclusions. This, as is the author's design, is left for the reader.

W. T. L.

PROGRESSIVE MEDICINE. A Quarterly Digest of Advances, Discoveries, and Improvements in the Medical and Surgical Sciences. Edited by H. A. HARE, M.D., assisted by H. R. M. LANDIS, M.D. Vol. VIII, June, 1906. Philadelphia: Lea Brothers & Co., 1906.

THIS volume is in keeping with the excellence of those previously noticed, the most important contributions to the literature being abstracted by men qualified to judge their value. Dr. Wm. B. Coley discusses Hernia and devotes several pages to an interesting defence of the Bassini operation. Dr. E. M. Foote summarizes the literature upon the Surgery of the Abdomen. Considerable space is devoted to the drainage of the peritoneum, surgery of the stomach, the appendix, gall-bladder, etc. One is impressed by the constant reiteration of the necessity for an earlier diagnosis of diseases of the stomach and yet nothing of importance seems to have been accomplished clinically toward furthering such a desire. Gastroenterostomy still remains the operation of choice; the various

papers by the Mayos are extensively abstracted. There does not appear to be anything new in the literature upon appendicitis. The most important summary of the surgery of the pancreas which has yet appeared, that of Villar, is abstracted. Dr. J.G. Clark, discusses Gynecology, opening his article with an interesting and comprehensive review of the recent literature upon the etiology of carcinoma. The review is almost entirely confined to the various diseases and malformations of the uretus. Dr. Alfred Stengel writes seventy pages upon Diseases of the Blood, discusses diabetes, gout, hemophilia, Hodgkin's disease, and concludes with that most interesting disease, exophthalmic goitre. Dr. Edward Jackson concludes the volume, writing upon Ophthalmology. Among a number of valuable abstracts, the bacteriology of conjunctivitis, and the causes of pterygium and of sympathetic inflammation are especially interesting. G. P. M.

WALTER REED AND YELLOW FEVER. By HOWARD A. KELLY, M.D., Professor of Gynecological Surgery in Johns Hopkins University, Baltimore. Pp. 293. New York: McClure, Phillips & Co., 1906.

To his other achievements Dr. Kelly has added that of an eminently successful biographer. His account of Dr. Reed's life and work is as sympathetic as it should be inspiring to younger members of the profession yet unknown to fame. Beginning with his birth and childhood, his college days, his graduation in medicine, his entrance into the army, his marriage, his frontier life, we are led to the beginning of his scientific work at the Johns Hopkins University in 1890 to 1899, his later studies in connection with yellow fever and its propagation by the mosquito, and finally to the circumstances of his untimely death. Aside from the biography, which is replete with sympathy, appreciation, and admiration for the man and his work, Dr. Kelly furnishes an extremely interesting account of the history of yellow fever—from the earliest days, through the many epidemics and the speculative reasoning of Carlos Finlay relative to the relation of the mosquito thereto, to the final demonstration by Reed and his colleagues, Carroll, Lazear, and Agramonte, of the undoubted etiological significance of the mosquito in the spread of the infection. Of Reed, Dr. Kelly says: "Though a man of war, he ravaged no distant lands, he destroyed no tens of thousands to make his reputation, but by quiet methods, when there was no strife, he saved countless lives and swept away a hideous plague, which from time immemorial has periodically visited our shores, devastated our fair land, and too often snatched from the years of peace and plenty all their blessings." Such are the plaudits of the populace that his work is valued least by those

whom it has helped most; but he doubtless found his reward when he wrote to his wife: "The prayer that has been mine for twenty years, that I might be permitted in some way, or at some time, to do something to alleviate human suffering, has been granted." The book as an admirable biography, a pleasing and interesting narrative, and a record of magnificent achievement, forms a notable addition to contemporaneous literature. A. K.

THE PROCEEDINGS OF THE CHARAKA CLUB. Volume II. Pp. 152. New York: William Wood & Co., 1906.

THE Charaka Club, named for the Hindu sage, is a small and rather unique organization of medical men interested in the literary, artistic, and historical aspects of medicine. Volume II of their proceedings consists of an interesting collections of poems, essays, reminiscences, and a story, of which the following are of more than ephemeral value: Books and the Man, by S. Weir Mitchell; Fracastorius, by William Osler; Charaka and His Times, by Smith Ely Jelliffe; Self-mutilation of the Eyes by an Ancient Saint and a Modern Sinner, by Ward A. Holden; The Medicine of Horace, by Charles L. Dana; and the Diseases of Learned Men, by Walter B. James. A. K.

INFECTION, IMMUNITY, AND SERUM THERAPY. By H. T. RICKETTS, M.D., Instructor in Pathology in the University of Chicago. Pp. 600. Chicago: American Medical Association Press, 1906.

THIS volume consists of the reissue in book form of a series of articles originally published in the *Journal of the American Medical Association*. It is divided into two parts, of which the first comprises a discussion of infection, immunity, toxins, antitoxins, and serum therapy, as well as the related subjects of agglutination, precipitation, phagocytosis, hemolysis, etc.; and the second part, of a more or less detailed discussion of the different infections that affect man and the lower animals. The book is a good and judicious summary of existing knowledge of the subjects of which it treats, and forms a valuable addition to medical literature. A. K.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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Gallop Rhythm of the Heart.—FRIEDREICH MÜLLER (*Münch. med. Woch.*, 1906, liii, 785). Gallop rhythm of the heart consists in the interposition of a third sound in the cardiac cycle. This sound occurs in diastole and is associated with a definite shock which is visible and easily palpable and makes a marked impression on the cardiographic record. In the cardiogram this impression may appear immediately before the systolic rise—the presystolic type, or in the first half of diastole, shortly after the second sound—the proto-diastolic type. Both of these waves are visible in the record of a normal apex-beat, although they are then very small elevations. They are pictured and studied by Marey in his classical work on the circulation and are particularly well illustrated in the tracings of Edgrem. It has been noted, too, that in cases of acute pericarditis in which there is certainly no change in the heart muscle, that the friction rub has a triple rhythm entirely analogous to the gallop rhythm. The occurrence of the first or proto-diastolic wave corresponds with the period of greatest relaxation of the ventricular muscle and is synchronous with the negative wave or drop in the jugular pulse tracing; it marks the time when the blood begins to flow from the auricle into the ventricle. The presystolic elevation comes immediately before the ventricular contraction, is synchronous with an elevation in the jugular tracing, and is due according to Marey, not to the auricular contraction, but to the increased velocity of the blood flow from this contraction.

The numerous curves which Müller presents show the justice of Potain's division of gallop rhythm into the presystolic and the proto-

diastolic type. In some curves the third sound occurs just before the systole and is marked on the cardiogram by an accentuation of the normal pre-systolic elevation, and occurs synchronously with a rise in the jugular pulse. The occurrence of this presystolic sound might be due to an unusually large amount of blood being thrown into the ventricle or to a loss of tone and elasticity in the ventricular wall. Potain assumes that a relaxed or sclerosed ventricular wall lends itself more easily to dilatation and oscillation (erschütterung) than a normal one, and he gives his support to the second view. Although such a notion is purely hypothetical it draws some support from the observation that dilatation is nearly always present when there is gallop rhythm. Chauveau, who favors the other view, insists that since gallop rhythm is so frequently a transient feature its occurrence cannot depend upon a permanent change in the ventricular wall. That the presystolic sound, on the other hand, is due to an exaggerated activity of the auricle, is suggested by the frequent finding postmortem of dilatation of the auricle and a considerable hypertrophy of its wall. Such a finding, however, is not constant. Frank has shown that the pressure increase caused by the auricular contraction when the auricle is better filled and more distended is plainly shown in the ventricular curve, and Marey, too, points out that the presystolic wave in the cardiogram is more marked when the auricle discharges its blood into a ventricle already filled, or in other words, a ventricle incompletely emptied. The evidence indicates that the presystolic gallop rhythm depends upon an increased discharge of blood from the auricle into the ventricle. It is interesting to note that, as a rule, in presystolic gallop rhythm the presystolic wave is further away from the ventricular systole than normal—that is, the time between the auricular and the ventricular contraction is increased. This might be explained upon the characteristics of the heart muscle as brought out by Englemann, namely, the retarded conductivity in dying muscle or in damage to the auriculoventricular bundle. Or one might think that when the ventricle empties itself only incompletely a greater tension is required in the auricle to overcome the resistance of an already filled ventricle and that the auriclar contraction is thus prolonged, just as the ventricular systole is prolonged in the face of increased arterial tension.

Müller also shows curves of typical proto-diastolic gallop rhythm in which the elevation occurs early in diastole and is synchronous with a venous negative pulse. In many of these tracings the presystolic elevation is likewise well-marked, and the close relation between the two types is further indicated by the ease with which one passes into the other. It is frequently noted that as conditions grow worse an existing presystolic gallop rhythm will change to a proto-diastolic. Barié insists that the latter is the more serious form and that it is always associated with dilatation of the ventricle. The time in the cardiac cycle at which the proto-diastolic sound occurs, the fact that the two waves appear so frequently in the same curve, and that the two sounds so repeatedly change from one to the other, lead one to assume a similar cause for both, namely, an increased flow of blood from the overdistended auricle into the ventricle. As a general rule, the presystolic type occurs with a slow, forcible heart action, the proto-diastolic when the heart action is more seriously disturbed, as evidenced by rapidity and irregularity and signs of circulatory stasis.

Another feature in Müller's curves is the relative length of systole as compared with diastole. This lengthening of systole is apparently due to a delayed relaxation of the ventricular wall which requires a more rapid outflow of the auricular blood when relaxation is complete. As Barie has observed, gallop rhythm occurs principally in conditions in which the blood pressure is high, but the fact that it occurs, too, in infectious diseases in which the pressure is low and in which there may be evidence of disease of the heart muscle, and that in cases of nephritis and arteriosclerosis and myocarditis it is not infrequently present at a time when the pressure is low, indicates that high tension is not the all-important factor that Barie considers it. The condition depends more probably upon the disturbance of the adjustment between the heart's strength and the resistance to be overcome. Gallop rhythm is found almost always associated with evident signs of cardiac insufficiency, no matter whether this insufficiency be due to an increase in peripheral resistance or decrease of cardiac power. As the circulatory conditions become readjusted the gallop rhythm tends to disappear. That the ventricle is in some instances struggling against a resistance which it can overcome only with great difficulty is indicated in some curves by a definite prolongation of the time of closure or the time between the beginning of the ventricular contraction and the opening of the semilunar valves.

Besides the increased resistance and the lessened cardiac power the rapidity of the heart-rate is also of importance in causing gallop rhythm. Schiff was able to produce gallop rhythm in dogs by cutting the vagus. In Basedow's disease and in tumultuous heart action from any cause the same tendency to gallop rhythm is noticed. In these cases it is due probably to the very short duration of diastole. The systoles crowding in one upon another, the auriculoventricular bundle has not time to recover its conductivity, and the distance between the auricular and the ventricular beats becomes so great that it may almost seem that the auricle is beating after the ventricle.

The prognosis of gallop rhythm is not always bad. Many patients live comfortably for years after its onset. Its occurrence, however, should always demand our earnest attention, and it is frequently an indication for active therapeutic measures, particularly for the administration of digitalis.

Gout and its Causation.—HALL (*The Practitioner*, 1906, lxxvi, 361) discusses the causation and treatment of gout and reviews the recent literature on the subject. His own work on the metabolism of gout is so well known and trustworthy that the following paragraph may be appropriately quoted:

"Uric acid cannot be regarded as a distinct factor in the causation of disease. We have still much to learn concerning its circulation in the tissues, and its capabilities for combinations therein. We need also further information as to its manner of excretion from the human body, and its varying behavior in individuals presenting different metabolic powers. Under properly arranged conditions, the rate and extent of its excretion may be utilized for the interpretation of the personal tolerance for exogenous nucleins, and for the elucidation of some of the intermediary stages in metabolism; but as an etiological entity, uric acid must be definitely discarded. It is high time that every practitioner made a point of fully educating the public in this subject."

The bacterial origin of gout is discussed. Trautner regards Bacillus coli communis as the cause of the disease. Others have supported the bacterial theory. Regarding the subject Hall says: "We cannot yet suppose a specific organism for gout, and the general metabolism plays a greater part in regard to the action of accidental or exceptonal intestinal toxins than is generally acknowledged. At the same time, it must be conceded that hereditary deficiencies in digestive faculties, or idiosyncrasies of metabolic functions, may permit the formation of exceptional toxins, or decrease the resistance to products of the ordinary intestinal bacteria."

Much work has been done on the relationship of glycocol to the etiology of gout. Hall, himself, finds it present in the urine in 70 per cent. of the cases, but states that there is no present proof that it is a causative factor. Shearer has suggested that an excessive quantity of carbonic acid in the arterial blood is a condition that leads to gout, and states that exercise, consequently, has a beneficial effect upon those subject to the disease because it increases the exhalation of carbonic acid.

Hall claims that hepatic insufficiency accounts for many of the symptoms of gout. Hepatic stimulants give the best results. He does not state what ones he uses. The accurate adjustment of food to the capacities of the intestinal mucosa and the liver is one of the best forms of treatment. The purins in the food and the purins in the urine are taken as the indicators.

Bacteriology of a "Common Cold."—BENHAM (*Brit. Med. Jour.*, 1906, ii, 1023) made a bacteriological study of the nasal secretions in twenty-one cases of mild "common cold" occurring in the vicinity of Brighton, England. There was no influenza epidemic prevailing. The clinical symptoms were usually sore throat, sneezing, malaise, headache, more or less nasal discharge, and slight fever. Most of the patients were able to go about. The previous work done on the bacteriology of the nasal discharge in normal persons and in persons with colds is reviewed. Of the 21 cases diphtheroid organisms were found in 20 (95 per cent.) of the cases. In 5 these bacilli were isolated in pure culture. Cocci negative to Gram's stain were found in 10 cases (48 per cent.) and isolated in pure culture in 2. Other cocci positive to Gram's stain were seen in 14 cases (67 per cent.). Some were undoubtedly pneumococci, but they were few in all cases and not isolated in any of them. Pfeiffer's influenza bacillus was found in only 2 instances, but was not isolated in pure culture.

It will be seen that a diphtheroid organism was almost a constant accompaniment of the colds. The cultural and biochemical characteristics of the organism are described in detail. Microscopically it is a short bacillus which tapers to rounded ends, In early cultures it is so short as to resemble an oval coccus. The average length in a three-day-culture is 1.5 microns and the breadth 0.3 to 0.4 micron. By the third day a characteristic appearance develops. The protoplasm at the ends of the bacillus is deeply stained, leaving an unstained band or septum across the middle of the bacillus. With a good illumination the outline of the bacillus can be clearly seen, especially in specimens counterstained with carbol-fuchsin. Benham thinks the organism has frequently been mistaken by previous observers for a diplococcus or

diplobacillus. Cultural and microscopic characteristics are cited that distinguish the organism from the true diphtheria bacillus.

The pathogenicity of the organism in animals was not definitely determined. Benham has suggested the tentative name of Bacillus septus for the bacillus. He thinks that it is probably identical with the organism isolated by Cantley and called Bacillus coryzæ segmentosus. He believes that the organism is responsible more for the sore throat, headache, malaise, and muscular pains than for the coryza. The coryza, when present, was probably due to the micrococci found and these he stated had the characteristics of the Micrococcus catarrhalis of Pfeiffer. The investigation favors the view that "common colds" are in all probability due to bacterial infection.

Xanthoma Tuberosum Diabeticorum. ABRAHAMS (*The Post-Graduate*, 1906, xxi, 533) reports an instance of this form of xanthoma of eight years standing in a male diabetic, fifty years of age. It began as a small, painful nodule at the nape of the neck near the border of the hair. After the patient first picked it off with the finger nail, it returned and steadily grew. One year after its appearance diabetic symptoms became marked. The mass gradually grew until it reached an inch by an inch and a half in size. It was made up of a series of nodules or tubercules of hazel-nut size—round, smooth, and lobulated. The upper border of the growth was red, the lower border pale.

The patient eventually began to experience difficulty in swallowing. He was unable to swallow any solid food whatever. The passage of a bougie revealed a stricture of the œsophagus near the cardiac orifice of the stomach. Two well-known clinicians, overlooking the xanthoma on the neck, made a diagnosis of carcinoma of the œsophagus. With proper diet and medicines the patient became aglycosuric, the tumor steadily diminished in size, and the dysphagia gradually disappeared, so that he could eat chopped meat, potatoes, and crackers. Xanthomata are well known to attack the œsophagus, heart, and other internal organs in diabetes, and Abrahams thinks that there is little doubt that the stricture of the œsophagus in this case was due to a local xanthomatous tumor. It is well known that xanthomata in diabetes disappear with the disappearance of the sugar in the urine, to return again with the reappearance of glycosuria.

On the Presence of Kernig's Sign in Zona.—BELBEZE (*Archives Générales de Médecine*, 1906, lxxxiii, 520) states that in the last four years he has observed nineteen cases of herpes zoster or zona, and has examined each case for the presence or absence of Kernig's sign. In only two instances was it present. Both cases were in women, one sixty and the other sixty-five years of age. In both instances also the eruption was along the course of one or more branches of the lumbar nerves.

Complications of Acute Pneumonia.—HENRY JACKSON (*Boston Med. and Surg. Jour.*, May 31, 1906, p. 620) has analyzed the various complications occurring among 1320 cases of acute lobar pneumonia treated at the Boston City Hospital. The complications directly connected with the lung were as follows: Empyema, 22 cases; lung abscess, 4 cases; lung gangrene, 3 cases; pleurisy with large effusion, 23 cases; unresolved pneumonia, 5 cases.

The complications not directly connected with the lung were: Pericarditis, 22 cases; phlebitis, 6 cases; meningitis, 3 cases; joint disease, 1 case; neuritis, 2 cases.

These complications probably fall considerably short of the actual total, for they include only the clinical diagnosis, the autopsy records not having been included in the analysis.

SURGERY.

UNDER THE CHARGE OF

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Simple Fractures of the Upper End of the Radius.—PRAT (*Revue d'Orthopedie*, March 1, 1906) says that it is astonishing how fracture of the upper end of the radius has passed without recognition, and how rare and brief are the descriptions of it in the books. He refers to about twenty cases from the literature, and reports four new cases. His conclusions concerning this fracture are based upon a study of the literature and his own cases.

The etiology, he says, requires no special mention. The usual cause is a fall on the hand, the force being received by the thenar side of the palm. Exceptionally the fracture may be due to direct violence, especially when applied to the posterior surface of the head, which is superficial. Quoting Tixier, he says that very rarely fracture of the neck may be due to violent contraction of the biceps muscle, the head being resisted by the annular ligament above. In the absence of any ligamentous attachment to the head or neck of the radius, fracture by the tearing of a ligament cannot occur.

Most frequently the head or neck is broken by a bending or crushing effect, the radius being caught between the humerus and the ground. The head does not continue the axis of the shaft, but forms an angle with it opening externally, so that a bending effect is produced in a fall on the hand. When this flexion does not occur the head receives the whole force and is crushed by the humeral condyle. The results of experimental fractures here, as in other parts of the body, are not convincing.

These fractures are divided into those which are complicated by other lesions of the elbow, and those which are uncomplicated. Fractures of the head are usually due to a crushing force, the lines of fracture being vertical or oblique. They are always intra-articular, making possible all the sequels consecutive to hemarthrosis. When a fragment of the head is rendered movable, it may become an intra-articular foreign body.

Fractures of the neck, when not due to an extension of a fracture of the head, are transverse or oblique. Prat believes that these are the cases due to a bending of the head downward. The horizontal line of fracture may be incomplete in some cases, that is, it may involve only a part of the thickness of the bone; in others, it may be complete and cause a decapitation. As a general rule the upper fragment is displaced backward and outward so as to lose contact with the inferior fragment, which is pulled upward and forward by the pull of the biceps. Sometimes there is a corresponding injury of the humeral condyle.

The symptoms will vary according to the type of fracture. In a simple fissured fracture the diagnosis will be confused with that of a sprain or contusion. The most characteristic sign will be the fixed and limited pain over the radial head, which rotates normally under the finger. In fracture of the neck with displacement, the symptomatology is more striking. When the fracture is complicated by neighboring fractures or dislocations, it may pass unrecognized. The skiagram, alone, can be positive in all cases. In fractures of the neck crepitus can be completely absent when the fragments are no longer in contact with each other, but the characteristic displacement will be present. This type may be confused with a luxation of the radius, the most characteristic differential sign being the rotation of the radial head in luxation, and its absence in fracture.

In simple fissured fractures without displacement, the prognosis is good for a full and rapid re-establishment of function, although the reactionary thickening may lead to future limitation of motion. The prognosis is grave in the fractures with displacement abandoned to a spontaneous cure. Consolidation of the fragments occurs in a vicious position. They may unite with the coronoid process, produce exuberant osteophytes, lead to false union, or to foreign bodies in the joint.

Prat says that a simple fissured fracture should not be immobilized. When there is a displacement of the upper fragment, its reduction is frequently impossible and when accomplished it is maintained with difficulty, so that there is only one recourse, excision of the head. This should not be done until the acute symptoms following the fracture have subsided, since the parts are then too sensitive to infection. The functional results were good in one case operated on, and in a second the patient was not seen after the twelfth day, when he was discharged with the wound cicatrized. Skiagrams later showed that some regeneration of the head had occurred in the first case.

Thomas (*Univ. Penna. Med. Bull.*, September-October, 1905) published a paper on this subject, in which he reported forty-eight cases from the literature, five new cases, and fifty-five from skiagraphic collections in Philadelphia, his own cases, however, being included in the latter. He produced on the cadaver, in five specimens, what he believes to be the typical vertical fracture of the radial head, by the same kind of force which is probably responsible for it in life, failing only in one attempt, and in that the occurrence of a fracture of the lower end of the radius was responsible for the failure.

His conclusions as to the mechanism, symptomatology, diagnosis and prognosis agree, in the main, with those of Prat. The latter, however, while calling attention to the remarkable frequency with which the fracture has passed unrecognized, throws no light on the

actual frequency of the fracture. Thomas has produced statistics which tend to show that it is as frequent as fractures of the patella. The axis of the head and neck can hardly be said to be at an angle with each other, as Prat says, since the upper cup-shaped surface of the head is at right angles with the axis of the shaft. One portion of the circumference of the head, however, is more prominent than the rest, and it is that portion which usually receives the brunt of the force in a fall on the hand, since it is anterior in pronation, and in extension of the elbow the humeral condyle is in contact only with the anterior portion of the radial cup. Prat has not recognized this fact, which was pointed out by v. Bruns, in 1881. It is just this portion of the head which is broken off in the usual vertical fracture of the head, and driven toward the palm in transverse fractures of the neck, according to Thomas' investigations.

The most confusing feature associated with this fracture is the change in position of the fragment or fragments by rotation of the forearm, the head normally rotating to the extent of about 180 degrees. When Prat says that, as a general rule, the displacement of the upper fragment is posteriorly and externally, without describing the position of the forearm, the statement has little meaning.

Those cases in which the absence of crepitus is due to so great a pull of the biceps on the lower fragment that the fractured surfaces are no longer in contact, are probably much more rare than Prat states. Some impaction is probably associated with the bending of which he speaks, in many cases, as it is in fracture at the lower end of this bone, and from the same cause. In two of Thomas' cases it was evidently present, and in Mütter's probably so. It would account for the difficulty in reducing the deformity and for the absence of crepitus. In view of the excellent results which followed non-operative treatment in one of Thomas' cases, and practically no treatment at all in the other, this being examined long after it was well, the statement that fractures of the neck with displacement of the upper fragment, which is probably always present, indicates an excision of this fragment in every case, can hardly be considered good surgery. Such a rule would very likely lead to many unnecessary infections of the elbow-joint.

Prat has very little to say of the vertical fractures of the head, which are probably much more frequent than the transverse fractures of the neck. His statement that the skiagram is always to be relied on for the diagnosis of these fractures, will not hold true in this type. Thomas reported one case in which a lateral view showed the fracture distinctly, while the anteroposterior gave no sign of it, although both skiagrams were equally clear. The frequent close approximation of the fragments by the orbicular ligament, the difficulty in determining the line of fracture, and the frequency of obscure shadows, present difficulties that are not to be overcome without great care.—J. W. W.

The Technique of Gastrojejunostomy.—Mayo (*Annals of Surgery*, April, 1906) states that the results of this operation depend more upon the condition of the patient when operated on than on the technical difficulties of the operation. The serious cases of acute "vicious circle" occurring during the first week, have practically disappeared with the evolvement of better methods. The most common subsequent complication is the chronic regurgitation of bile, which comes on at

intervals in a small percentage of cases. Since January 1, 1905, all "loop operations," with or without enteroanastomosis or closure of the pylorus, have been discarded, the gastric anastomosis being made as close to the origin of the jejunum as possible. Of fifty-six cases operated on in this way, two developed chronic biliary regurgitation of a serious character. Re-operation in both showed that the cause was an angulation of the jejunum at its gastric attachment, due to partial twisting as a result of suturing in the line of peristalsis. Since July 1, 1905, the reversing of the jejunum has been abandoned, and in a larger number of cases, sixty-five, not a single case gave trouble and no death has occurred. The jejunum is applied to the posterior wall of the stomach from right to left exactly as the intestine lies in the abdomen. The idea of reversing the peristalsis is not original with Mayo.

Traumatic Defects of the Skull; Their Relation to Epilepsy; A Clinical and Experimental Study of Their Repair.—ALLEN, SANFORD, and DOLLEY (*Boston Medical and Surgical Journal*, April 12, 1906) report seven cases in which traumatic defects in the skull were repaired by the implantation of grafts taken from another part of the skull, that consisted of an equal-sized area of pericranium and attached external layer of the skull. In the removal the bone necessarily was broken into small fragments. Any connective tissue covering the exposed portion of the brain is carefully removed. Since the scar tissue and dura are often inseparable the pia covering the brain may be thus exposed. The graft is carefully laid in position and covered with the scalp, a cigarette drain being employed for a few days. Healing by first intention occurred in every case, and the defect was repaired by what appeared to be bony structure. A macroscopic and microscopic study was made in a series of successful experimental grafts made on dogs.

Non-lithogenous Obstruction of the Biliary Ducts.—CORDIER (*Surgery, Gynecology, and Obstetrics*, April, 1906) reports one case in which the obstruction was due to an indurated mass just where the common duct passed behind the duodenum. This was the site where a large stone had ulcerated into the duodenum, and in healing had formed a complete stricture of the duct. The gall-bladder held four pints of bile and mucus, but no stone was found after a careful search of the ducts. An anastomosis was made between the gall-bladder and jejunum. The symptoms later disappeared entirely and three years after operation the patient was entirely well.

Strictures of the cystic duct are more likely to form than in the common duct. The surrounding structures are more dense and unyielding; there is no constant flow of bile to wash away infection from the intestine, or any sedimentary nucleus; and when once formed an obstruction tends to become complete. In such a case, cholecystostomy with drainage may be sufficient for treatment. With the subsidence of the acute symptoms, the canal becomes pervious, and bile begins to flow freely from the established fistula, when the end has been attained. It is better, however, when the gall-bladder is full of strictures to remove it, as in the case of the appendix. In the chronic cases, when the gall-bladder is a mass of scar tissue, a cholecystectomy should be done.

In a second case, a man, aged twenty-one years, had had attacks

of "colic" since he was three years old. At operation, just where the common duct passed under the first part of the duodenum, the duct was as hard as a tendon, and appeared to be in very much the same pathological condition that is found in appendicitis obliterans (cholangitis obliterans). Cholecystojejunostomy was again performed with a Murphy button. Satisfactory cure resulted, which Cordier is afraid will not be permanent, owing to the change produced in the liver and spleen by the long-continued (eighteen years) obstruction. In a third case a similar condition resulted in the common duct, from the kick of a horse, and was cured by a similar operation. In a fourth case the cause was a chronic pancreatitis involving the ductal opening into the duodenum. One year after the performance of the operation the symptoms had not recurred. Cancerous invasion here is frequently due to the existence for a long time previously of some benign condition. Cancer of the bile passages occurs most frequently in the lower portion of the common duct, the location of most lithogenous obstructions.

Treatment of Varicose Veins.—Mayo (*Surgery, Gynecology, and Obstetrics*, April, 1906) thinks that the most common etiological condition is the existence of a congenital defect in the vein walls or valves, or in the innervation. There is a considerable question whether normal veins give way under the ordinary strain of life.

One hundred and eight-five operations have been done for varicose veins, many upon both limbs. The complete Schede operation has not been performed for several years; but a partial Schede, or oval or interrupted incision above or about a large ulcer, is frequently done, and combined with excision of the ulcer and skin grafting. The Trendelenburg operation is also done. Mayo has been partial to the excision of the long saphenous vein in the majority of cases. This is done by long incisions, leaving five or six inches of skin intact at the thigh, a pair of forceps being employed to enucleate and loosen the vein in the uncut area.

More recently he has employed a ring vein enucleator, which consists of a quarter-inch ring of steel with a long handle, the whole instrument being not unlike a blunt uterine curette bent to an angle near its tip. He has also had made for the same purpose a pair of long forceps, which form a ring at the end when closed. Beginning the operation above, the vein is exposed, ligated, and cut. The lower end of the vein is passed through the ring of the enucleator or the forceps. In this way by a gentle pushing force the vein is stripped from the surrounding tissues and the lateral branches torn off for six or eight inches, when a small incision is made in the skin and the isolated vein pushed through. The process is repeated downward in a similar manner.

Calculous deposits, sacculations, or extreme weakness of the vessel walls, render the case unsuited for this method, and this occurs in about 10 per cent. of the cases. Hemorrhage is prevented by elevation of the leg on an ordinary gynecological standard. This renders the limb partially bloodless and secures accessibility of the field of operation. More than ordinary hemorrhage from torn branches can be checked by pressure with a pad.

THERAPEUTICS.

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Sodium Citrate in Infantile Vomiting.—M. VARIOT considers that sodium citrate is a very potent remedy in the treatment of the digestive disorders of infancy. Its employment is based upon the fact that when taken into the stomach it reduces the tendency of the milk to clot in this organ, and in consequence, increases its digestibility. Diarrhœal conditions and vomiting are especially amenable to treatment by sodium citrate. The author prescribes a solution of the salt in water, 5 parts to 300, of which he gives 1 to 2 drams before each feeding. The theory that the sodium citrate increases the normal citric acid of the milk is false; indeed, sterilization of the milk destroys the normal acid, and it is on account of the absence of this ingredient in sterilized milk, according to certain observers, that scurvy occurs in infants thus fed. Consequently, if this hypothesis be true, sodium citrate may be an efficient antiscorbutic.—*La Quinzaine Therapeutique*, 1906, No. 23, p. 536.

The Treatment of Mucous Colitis.—DR. R. SAINT-PHILIPPE reports excellent results from the following treatment: 1 to 5 minims of aconite tincture and 5 to 30 minims of syrup of ipecac (the dose being regulated in accordance with the age of the patient), mixed with a neutral vehicle, are given every two hours, day and night. If fever is absent the aconite should be omitted. The patient should be kept continually under the influence of the medication and no apprehension need be felt if constipation is induced. When improvement without complete recovery is noted, the author advises the substitution for the ipecac of a cold maceration of freshly powdered guarana, the dose of which is 7 to 15 grains in sweetened water during the twenty-four hours. Guarana is a sovereign remedy in dysentery after the acute stage is over and the fever has disappeared. The cause of the disease should, if ascertainable, be suppressed, fats should be interdicted, the drinking water should be of the purest, and fruits should be taken with care.—*Revue Francaise de Médecine et de Chirurgie*, 1906, No. 2, p. 27.

The Dietetics of Gastric Ulcer.—DR. SENATOR advises a diet feeble in nitrogenous elements, but which contains gelatin (the latter being both a hemostatic and a food), fat, and sugar. The consistence of this diet is fluid, it is sufficiently nourishing, and is non-irritant. It is prepared by dissolving 4 to 6 drams of pure white gelatin in about 6 ounces

of water to which have been added about 7 drams of citron oleosaccharide. This quantity is taken in twenty-four hours, together with 8 ounces of cream and 1 ounce of butter. The daily ration represents about 900 to 1000 calories. The gelatin may be replaced by analogous substances, such as calf's foot jelly or fish gelatin, and for the butter one may substitute olive oil, milk of almonds, etc., according to the tastes of the patient. To some patients it may be necessary after a week of this diet to give finely minced raw meat, others can bear the gelatinous diet for a longer period.—*La Semaine médicale*, 1906, No. 3, p. 33.

The Renaissance in Therapeutics.—Dr. R. C. Cabot summarizes a paper as follows: 1. Therapeutics has become, within the past few years, very noticeably more effective. 2. This increased effectiveness is the result of (*a*) the aggressive spirit as exemplified by the New York Board of Health in the School of Hygiene and Inspection, and by the tuberculosis work now being carried on in Boston; (*b*) the greater part now being taken by the laity in medicinal work, both by intelligent co-operation and by financial support; (*c*) our greater acquaintance with physical therapeutics, especially with the details upon which their successful application depends; (*d*) the rise of scientific mind cure and of social work, marking a recognition of the psychical and of the social elements in all diseases; (*e*) a wider utilization of the unique talents of women in the field of therapeutics. 3. With the rise of the type of therapeutics here described we are now witnessing a limitation of the sphere of surgical therapeutics and of drug therapeutics. The latter limitation will do much to undermine the superstitions on which the "patent medicine" habit rests.—*Journal of the American Medical Association*, 1906, No. 22, p. 1660. (It is gratifying that the therapeutic awakening which took place many years ago and in which the American Therapeutic Society had a prominent part is beginning to be recognized in some quarters. The broader concept of therapeutics will doubtless lead to still greater achievements in the healing art.—R. W. W.)

Cocillana as an Expectorant in Pulmonary Tuberculosis.—Dr. George W. Norris has employed this drug in thirty-one tuberculous patients in which an expectorant was indicated. The preparation used was a fluidextract of the bark of Syncocarpus rusbyi, a large tree found in Bolivia. The physiological action of the drug is said to resemble that of ipecacuanha, but as an expectorant it is supposed to possess a more stimulant effect than the latter, and also to act as a mild laxative and cardiac tonic. The author obtained the most satisfactory results from 5 drop doses given in water at three-hour intervals. Of the patients who received the drug, six were in the early, twelve in the second, and thirteen in the advanced stage of the disease. The results were as follows: Marked improvement, seven; slight improvement, fourteen; no improvement, eight; apparently worse, two. Improvement when it occurred was commonly manifested by a lessening of the viscidity of the sputum and a corresponding increase in the ease of expectoration. Cough as a symptom was little affected. The quantity of the sputum was little modified; there was no apparent effect upon the pulse rate or heart action. In four instances the response to the physiological action of the drug was prompt and brilliant, but in the majority the results were not very striking. No means were ascertained by which

one could judge beforehand which patient would respond best to treatment. In conclusion, the author states that the results achieved were not as satisfactory as those obtained from an expectorant mixture containing ammonium chloride, glyceryl nitrate, aromatic spirit of ammonia, nux vomica, and elixir of calisaya.—*The Therapeutic Gazette*, 1906, No. 6, p. 370.

Physical Agents in Acute Articular Rheumatism.—Dr. A. Laqueur deprecates the present tendency to neglect local applications in acute rheumatism and to depend wholly upon the salicylates. In connection with the latter he employs compresses lightly wrung out in cold water; these are wrapped about the inflamed joints and are kept moist by allowing water to drip upon them from time to time; by this means, the local inflammation is effectually relieved. In case of complicating endocarditis cold is applied to the precordium thrice daily for an hour at a time. When the temperature has fallen to normal the salicylates are stopped and full baths at 95° to 100.4° F., lasting from ten to twenty minutes, are given; if endocarditis is present the bath should not exceed 98.6° F. nor last more than fifteen minutes; following the bath cold is applied to the precordial area. Local treatment in the form of hot air, passive congestion according to Bier's method, centripetal massage, etc., is prescribed. If the acuity of the inflammation resists the salicylates, hot baths, followed by sweating induced by wrapping the patient in hot, dry sheets, are indicated; if the pains are obstinate ichthyol is added to the bath.—*Berliner klinische Wochenschrift*, 1906, No. 11, p. 329.

Sodium Citrate in Infant Feeding.—Dr. W. H. Wynn emphasizes the advantages accruing from the addition of sodium citrate to the artificial foods of infants thus: The common cause of milk-dyspepsia is the density of the clot formed in the stomach; sodium citrate increases the digestibility of milk by lessening the amount of clot, consequently by its use, milk can be given in more concentrated form and overdilution prevented. There is no danger of rickets or scurvy. The chief indications are (1) for correcting milk-dyspepsia; (2) for weaning a healthy infant to cows' milk. The amount to be added to each feeding for a child of one month is 1 grain dissolved in a dram of water. The sodium citrate may be increased or diminished with the age, progress, and weight of the infant. The author considers the salt also useful in adults when plain milk is not well borne, as in gastric ulcer, gastritis, pneumonia, enteric fever, etc. It is useless in the severe gastroenteritis of infancy, fat dyspepsia, with impure or adulterated milk, and in primary infantile atrophy.—*The Birmingham Medical Review*, 1906, No. 39, p. 123.

The Serum Treatment of Enteric Fever.—M. Josias has employed Chantemesse's serum in the treatment of enteric fever in children, for the past four years. Previously he had used cold baths, with a mortality of from 10 to 12 per cent. The death-rate under treatment with serum injections and bathing has fallen to 3.8 per cent. in nearly two hundred instances of the disease. Of the five deaths which took place perforation was responsible once, meningitis once, pneumonia once, laryngitis with otitis and pulmonary congestion once, and intestinal hemorrhage once. Of complications in the patients who recovered, hemorrhage

occurred five times, otitis three times, phlebitis twice, nephritis once, and periostitis once. Relapses were few and mild. From his results Josias concludes that Chantemesse's antityphoid serum is harmless and that patients treated by it. in connection with baths, stand a better chance of recovery than those treated by hydrotherapy alone.—*Bulletin de l'Académie de Médecine*, 1906, No. 10, p. 301.

Radiotherapy and Diphtheria Antitoxin in Pernicious Anemia.—M. RENON reports a case of pernicious anemia in which marked cachexia, œdema, and diarrhœa were present, and in which the blood, when the patient was first seen, contained 880,000 red cells and 2000 leukocytes. Under arsenic the condition was aggravated rather than improved, but the application of the Röntgen rays rapidly caused an increase in the red corpuscles to 920,000. Injections of antidiphtheric serum associated with the radiotherapy were followed by a further increase to 2,545,000, an amelioration of the general condition, and an increase in body weight. Examination of the blood after the injections and the application of the rays showed that a stimulation of the hematopoietic system had taken place, as evidenced by the presence of an increased number of red cells, an eosinophilia, and a polychromatophilia. On the other hand, the presence in the blood of the products of the destruction of the leukocytes by the rays or of hemolysins (which result from the serum injections) seems to be a necessary factor in determining a reaction of the blood-making organs.—*La Semaine médicale*, 1906, No. 11, p. 128.

Passive Congestion in Joint Disease.—DR. H. F. HARTWELL cites ten instances of tuberculous and gonorrhœal joint affections in all but one of which rather remarkable results were obtained by Bier's method of producing passive congestion. The congestion was caused by the application of an Esmarch bandage just long enough to encircle the limb two or three times at the desired tension and provided with strap and buckle at either end. The bandage should be applied just above the affected joint and secured by the buckle when the desired degree of congestion has been obtained; the skin may be protected by a few turns of an ordinary bandage; and to avoid stasis of blood in parts where it is not needed the limb which lies peripherally to the infected area may be snugly bandaged. Care should be taken lest the congested limb become cold to the touch and that the patient is not made uncomfortable and unable to perform his ordinary duties. In tuberculous joints the stasis is not kept up more than an hour daily; in severe gonorrhœal arthritides the congestion must be continued for ten or twelve hours and, while the strap is off, the limb should be elevated to reduce the œdema which the constriction has produced. Upon the subsidence of acute symptoms massage and passive motion should be instituted. Congestion produces no benefit in chronic hydrops after acute inflammation. In general, Hartwell states that the duration of the stasis should depend upon the effect obtained If the patient observes that after an hour or two the pain is relieved and motion is increased, this length of time is sufficient, but if the symptoms soon return a longer period of congestion is necessary.—*Boston Medical and Surgical Journal*, 1905, No. 18, p. 483.

The Treatment of Insomnia.—WILLIAM BROADBENT (*The Practitioner*, 1906, No. 1, p. 1), in discussing this subject, states that one of the most frequent causes of insomnia is gastrointestinal derangement, especially when associated with flatulence, and while distention of any part of the digestive tract may give rise to dreams and broken rest, or definite wakefulness, it is gastric dyspepsia which gives nightmare, and dilatation of the stomach which produces the most obstinate sleeplessness; consequently, the treatment of insomnia due to these causes should be directed to the etiological factor and all digestive disorders should be rectified. The diet should be simplified and the stomach must never be distended. The amount of bread and of other farinaceous foods, of green vegetables, and of liquid with meals should be restricted, water, hot or cold being drunk every morning. One or two tablespoonfuls of brandy or whiskey in a claret glass of hot water as the only drink at luncheon and dinner will often be useful in dislodging gases. Treatment should be directed to the prevention of gastric or intestinal fermentation, for which purpose salol, naphthol, creosote, carbolic acid or sulphocarbolates may be given, not forgetting calomel or other mercurial, usually a very important consideration. In old persons vascular degeneration may have gone so far that no physiological state of the cerebral circulation conducing to sleep may be attainable; functional derangements, destruction of sleep, may be due to structural changes which cannot be modified; or an idiopathic condition of restlessness may be established; here, after palliatives have been tried, we may give sedatives without hesitation. Late in life there is no danger of inducing a drug habit, and a moderate dose of an opium preparation may conduce to the comfort of the patient and even prolong life. The chloral group is less to be relied upon than opium; the latter may be given nightly with colocynth and hyoscyamus to prevent constipation. In the young, caution is necessary; opium should be avoided and the patient should never be allowed access to sedatives at will. When the sleepless habit has become established sleep may be induced for three or four nights from time to time by one or other of the chloral group with a view toward breaking the habit, or if the patient's self-control is not to be relied upon, paraldehyde may be substituted. Broadbent believes that such drugs as sulphonal, trional, and veronal are doing incalculable harm.

The Physiological Action of Tea as a Beverage.—LAUDER BRUNTON (*Quarterly Journal of Inebriety*, 1906, No. 2, p. 19) sums up a paper with the following statements: Tea when properly prepared and taken in moderation is both useful and agreeable. When taken in too great quantity, or along with butchers' meat, when too strong, when infused too long, or still more when boiled and stewed, it is apt to produce digestive troubles. When taken in excess it may produce nervous symptoms of the most serious character, and facilitate, if it does not actually produce, mental degeneration.

Expectorants.—H. EICHHORST (*Deut. med. Woch.*, 1906, No. 17, p. 649) considers that the irritating cough of tracheitis may be best relieved by 10 drops, three or four times daily, of codeine phosphate 4½ grains and bitter almond water 2½ drams. If the attacks of coughing are associated with nausea or if the appetite is poor he prescribes a mixture

containing codeine phosphate 4½ grains, raspberry syrup 5 drams, phosphoric acid 7½ grains, water 6 ounces; of this the patient takes two teaspoonfuls three times a day. He does not employ narcotics, other than codeine, because of their unpleasant by-effects. These are especially harmful when the bronchial secretion is copious, because of the possibility of producing cyanosis. True expectorants should have a solvent action upon tenacious sputum; in this connection the author recommends the following: potassium iodide 45 grains, simple syrup 5 drams, to which powdered ipecac 7½ grains infused in 6 ounces of water is added; dose 2 drams every two hours. The depressant effects of apomorphine and pilocarpine contraindicate their employment, but a powder consisting of benzoic acid 4½ grains, camphor ¾ grain, and powdered sugar 3 grains, may prove useful. In chronic processes with fœtid expectoration the balsams are indicated, especially terebinthine and myrtol. In bronchial catarrh of the infectious type benzosol and benzoguaiacol are excellent, and the catarrhal inflammation may be lessened by the administration of 20 to 30 drops of the fluidextract of hydrastis four times daily. Steam inhalations also act as an expectorant and in certain instances rhythmic compression of the lower thorax will aid in the expectoration of tenacious mucus.

Marmorek's Serum by the Rectum in Tuberculosis.—M. PETIT (*Revue de Therapeutique*, 1906, No. 10, p. 337) has employed the following technique in the administration of this agent. Every two days, after a preliminary cleansing irrigation. 0.75 of a minim of the serum is injected, after which the patient rests for a time. Of twenty-seven patients so treated, seventeen have been improved and five no longer exhibit physical signs; five have died—all of whom were in the third stage of the disease. Under the treatment the first sign of amelioration is a disappearance of the dyspnœa, which generally takes place after about five injections; later the sputum disappears and finally the physical signs are modified. The general condition remains unchanged. Hemoptysis is not a contraindication, and the author has observed no unfavorable reaction of any kind; in his opinion the treatment seems specific and curative; at any rate it offers no disadvantages. In discussing the paper M. Le Gendre stated that the evidence presented could not, in his opinion, be considered seriously, since the cures or improvements had persisted not more than three or four months, and in consideration of the natural remissions which so often characterize the disease the results hardly constituted a sufficient proof of the efficacy of the treatment.

Thiocol in Non-tuberculous Pulmonary Affections.—M. NIGOUL (*Le Progrès médical*, 1906, No. 14, p. 215) considers that in thiocol we possess a very practical means of administering creosote in such affections as pneumonia, acute and chronic bronchitis, bronchiectasis, etc. The minimum dose, when the object is to drain the bronchi of the secretion, is 30 to 45 grains per day. A smaller dosage will suffice when it is not desirable to relieve the mucous membranes of inflammatory products. In a reported instance of emphysema, daily amounts of from 15 to 25 grains were sufficient to prevent exacerbations of bronchitis and pulmonary congestion. The drug also exercises an advantageous effect upon the digestive system which aids in augmenting the bodily resistance

PEDIATRICS.

UNDER THE CHARGE OF

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Early Diagnosis of Measles.—BRELET (*Archives Générales de Médecine*, 1906, lxxxiii, 669) discusses the different methods suggested to make a diagnosis of measles before the rash appears. During the period of incubation this is usually considered impossible. There are two signs during this period upon which some reliance can be placed. The first is Meunier's sign, a marked loss in the weight of the child; it is usually present and appears first about the third, fourth, or fifth day of incubation and eight to ten days before the eruption; the loss in weight averages about 60 grams per day; its cause is probably a disturbance of nutrition brought about by the measles infection. Meunier says the sign may be found in the incubation period of other infectious diseases as well, but considers it of value in cases in which children have been exposed to the infection; weighing them each day will show which of them is likely to develop measles. The second sign during this period is an increase in the number of leukocytes, especially the polynuclear forms. This leukocytosis, beginning with the period of incubation, increases rapidly and attains its maximum about the sixth day before the appearance of the rash; it then diminishes during the period of eruption and in cases of uncomplicated measles a leukopenia is present. At the period of invasion a number of signs have been described, which may assist in the diagnosis before the appearance of the rash, but the only one pathognomonic of the disease is Koplik's sign. It consists of rose-colored or bright-red spots having bluish-white centres; they are found on the mucous membrane of the cheeks and lips. The red spots, at first small and rounded, enlarge and may unite with the neighboring spots. The central point is the pathognomonic sign; it is very small, being from 0.2 to 0.6 mm. in diameter, is slightly raised and only removed from the surface with difficulty; the mucosa upon which the spot rests often presents a roughened appearance, which is very characteristic. Koplik's spots do not ulcerate; they disappear in three or four days. They may be few in number or very numerous, and never occur on the gums. They appear usually three days before the eruption and occur in from 80 to 100 per cent. of the cases. During the period of incubation, therefore, a diagnosis is rarely possible. At the stage of invasion, the presence of Koplik's spots, appearing as they do several days before the cutaneous rash, makes the diagnosis of measles certain.

Examination of Milk Bought in the Leipzig Market, with Special Reference to Streptococci.—H. BRUENING (*Jahrbuch f. kinderheilkunde*, 1906, lxii, 1–22) reports having examined 40 samples of milk

(12 sterilized, 26 raw full milk, 2 buttermilk) during the summer months of 1904. Of the samples of raw milk, 7 were amphoteric, and 19 alkaline; the fat maximum was 4.05 per cent., the minimum 2.19 per cent., the average amount being 3.12 per cent.; 5 samples did not reach the amount of fat prescribed by law (2.8 per cent.). The 12 samples of sterilized milk were all free of bacteria; of the other 28 samples, 26 (93 per cent.) contained streptococci in varying quantities; 2 samples had 100 organisms to the c.c., 6 had 1000, 8 had 10,000, 8 had 100,000, and 2 had 1,000,000. There were two varieties of streptococci: the first, Gram-positive, composed of thick individuals, a few making a chain; they produced death in white mice within twenty-four hours. A second group consisted of Gram-negative, narrow diplococci, as well as similarly arranged Gram-positive organisms, non-pathogenic to white mice and guinea-pigs. The streptococci were not present in milk immediately after milking, as proved by a number of samples taken directly from the animals; they entered the milk during transmission to market and are probably chiefly saprophytic. When fed to animals they were found to be absolutely innocuous, but as epidemics of dysentery have been reported in cases in which the only organisms found were these same non-pathogenic bacteria, these streptococci deserve attention at the hands of bacteriologists and specialists in children's diseases. A number of observers have discussed the possibility of a relation between them and the summer diarrhœas of children.

Bronchopneumonia and Pyemia following Measles.—J. McKENZIE (*Lancet*, 1906, i, 1315) reports the case of a child of twenty months, with a history of having been in good health and found suffering from an attack of measles associated with delirium. The rash appeared on the fourth day of the illness, and on that date, except for the delirium, there was nothing exceptional about the case. On the sixth and seventh days of the illness the rash had almost disappeared, but the temperature had not dropped to normal and the child was very restless and delirious. Evidence of commencing pulmonary consolidation could be detected at the left base, but the nervous symptoms were not such as one might expect at the beginning of an uncomplicated pneumonia with moderate temperature. No evidence of meningitis or osteomyelitis could be found. The delirium persisted until the end, which occurred on the eleventh day of the disease. The most prominent features were the persistent delirium with moderate temperature and the very rapid and advanced consolidation of the left pulmonary base. The autopsy revealed red hepatization of the lower left lobe and minute abscesses scattered over all the organs of the body. Bacteriological examination of the fluid from the lungs, spleen, pericardial and cerebrospinal cavities, showed the presence of Staphylococcus aureus. The case is curious because of the staphylococcus inducing a pulmonary consolidation; because of the rapidity with which the lower lobe became atelectatic; because of the virulence of the organism giving rise to embolic infection of the various internal organs; and because of the constitutional symptoms of the intoxication overshadowing the effects of the local pulmonary lesion.

Nervous Complications in the Typhoid Fever of Children.—G. B. ALLARIA (*Gazetta degli Ospedali*, 1906, xxxvii, 17) describes several cases of typhoid fever in children complicated with affections of the nervous

system. The first patient, a girl, aged six years, developed meningismus, right-sided spastic hemiparesis, and aphasia; the second, a boy, aged six years, developed in addition Jacksonian epilepsy. Both patients recovered completely. There was no cardiac or renal disease. In the first patient's case the right hemiparesis developed in the fourth week of the illness, was followed three days later by neck rigidity, and later by motor aphasia, which lasted three weeks. The second patient developed the meningeal symptoms during the second week, motor aphasia in the third week, and right-arm Jacksonian epilepsy in the fourth week. Lumbar puncture gave some temporary relief. The increased knee-jerks persisted on the right side; the aphasia lasted two weeks. A third patient, a girl, aged eight years, died as the result of a typhoid meningitis. Most of the typhoid paralyses, as seen in the first two cases, come on gradually, and have no relation to the gravity or intensity of the intestinal lesion. They are often accompanied by irregularities in temperature, perhaps due to irritation of the heat centres. The differential diagnosis between a toxic and organic paralysis is difficult, and all the more if there are symptoms of meningismus present, although these latter are rare in typhoid. The cerebrospinal fluid gives the best clue in their differentiation. The fourth patient had tetany complicating the typhoid.

Malignant Teratoma of the Ovary.—L. S. DUDGEON (*Jour. Obstet. and Gyn. Brit. Empire*, 1906, ix, 28) reports the case of a child, aged four years, who had always been well until within a few months; since then her abdomen had gradually enlarged. Examination showed the abdomen to be occupied by a large mass; its upper border reached the costal margin, its lower, the pelvic brim; fluctuation could be detected. The abdomen continued to increase in size, marked abdominal pains developed, the right foot became œdematous, and tapping removed a large quantity of thick, greenish fluid. She died five months after admission to the hospital; before death, numerous nodules appeared all over the abdomen and scattered hemorrhages covered the skin. The autopsy revealed a cystic tumor of the left ovary, filling the abdominal cavity, but not adherent anywhere; one secondary nodule was found in the liver. The surface of the tumor was irregular, with cystic and hard projections all over its external surface. Microscopic examination showed the tumor to be of the class of teratomas. An excellent microscopic description of the tumor is given, well illustrated by pictures. Dudgeon says that the earliest age at which such a condition has thus far been described is six years.

Lavage in Acute and Chronic Gastritis of Children.—E. L. COOLIDGE (*Bull. Lying-in Hosp., New York*, December, 1905, ii) prefers stomach washing to any of the treatments for acute and chronic gastritis in infants. One single washing often suffices in acute gastritis; in semichronic or chronic cases daily lavage may be necessary for a time. In addition, the food, bowels, and general hygiene of the infant's life must be rectified. A No. 16 catheter with a large eye is attached by means of glass tubing to a rubber tube, to which a 4 to 6 ounce glass funnel is fitted. Previously boiled water cooled to 100 to 110° is employed; a teaspoonful of bicarbonate of soda is added to the quart of water when much mucus exists. The child is held upright in the mother's lap;

a rubber sheet is tied around it. The tube is lubricated with olive oil, and passed rapidly through the pharynx and œsophagus into the stomach; from eight to ten inches of tube will thus be swallowed. Gas and food are permitted to escape, four to six ounces of water poured in, and the water, food, and mucus siphoned out. After the water comes away clear, the tube is removed. In chronic cases, or when there is much prostration, it is an excellent plan to leave in the stomach an ounce of fresh, hot water, together with a dram of a good liquid beef preparation. The lavage should be performed two and one-half to three hours after feeding and no food should be given for an hour afterward, the child during that time lying on its back. Two cases are reported in detail.

The Economic Production and Distribution of Clean Milk.—J. ROBY (*Jour. Amer. Med. Assoc.*, 1906, xlvi, 1430) demonstrates how remarkably clean milk can be produced without an expensive plant, citing the case of a farmer with but a cheap outfit. He counts up the steps necessary to produce such a milk, and lastly describes a scheme to improve the entire milk supply of large cities. The rules to produce clean milk are as follows: (1) It must be protected from infection by scarlet fever, diphtheria and typhoid by removing the patient from the farm; (2) it must be cooled at once and kept cold, or at least below 50°; (3) the utensils must be kept clean, or better still, sterile; (4) milking should be done through a sterile cheese-cloth into a pail with a small (five inch) opening; (5) the pail must be kept tightly covered. These five points are absolutely necessary to produce milk containing a low number of bacteria; if the rest of the points are obeyed, a milk containing probably no more than 1000 bacteria to the c.cm. will be produced. (6) Discard the first draw from each teat; (7) keep the cow absolutely clean; (8) keep the milker's hands and clothes clean; (9) keep the ceiling free from dust; preferably it should be lathed and plastered; (10) keep the barn clean and use shavings for bedding instead of straw; (11) dust should not be stirred just before or at time of milking. The scheme for improving the milk supply is, that the city have a can sterilizing plant. The cans must first be washed, the cheese-cloth put over them, then the metal cover, and lastly, a canvas cover with a purse-string to keep it free from dust. The cans are sterilized by dry heat, shipped back to the farmer, who milks directly through the cheese-cloth. He removes the cheese-cloth, replaces the cover and sinks the can into ice-water up to its neck. Then the whole outfit is returned to the city to be bottled or delivered directly. Milk thus handled is said by the author to contain less than 500 bacteria to the c.cm.

OBSTETRICS.

UNDER THE CHARGE OF

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Anesthesia during Labor by Scopolamine-morphine Injection.—GAUSS (*Archiv. f. Gyn.*, 1906, Band lxxviii, Heft 3) reports the results following the injection of scopolamine-morphine in 500 cases of labor

at the Clinic in Freiburg. These patients illustrated the various complications of labor, and among them the different obstetric operations were performed. The preparation employed was Merck's and the result of the injections so far as labor was concerned was satisfactory. Patients differed in the cerebral symptoms produced by these injections in accordance with the susceptibility of the nervous system. So far as the performance of obstetrical operations was concerned, in a few cases the use of forceps, suture of the perineum, and in one case hebotomy, were done without other anesthesia. In most cases, however, additional anesthesia was required. The patient's injection did not seem to be an unfavorable element in the use of another anesthetic. A solution of 0.03 per cent. of scopolamine and 1 per cent. of morphine in sterile distilled water was that most usually employed. In quantity a dose of 0.00045 to 0.0006 hydrobromate of scopolamine with 0.01 morphine was employed. If this failed to produce a result a second injection of 0.00015 to 0.0003 scopolamine was given without morphine. If in from two to four hours no result followed, then 0.005 morphine was added. The time required to produce an effect varied from one-half hour to fifty-seven hours. The majority of cases, however, experienced marked relief within two to six hours after the injection. The longest time through which the effect endured was forty-eight hours, during which a patient was treated in this manner. The results of this treatment are analyzed in detail, as regards the mother's general condition, labor pains, abdominal pressure, length of labor, necessity for operation, and the effect upon the child. It was impossible to trace injurious effects, in any of these particulars, to the injection. The conclusion is reached that the use of this material is without injurious effect, does not interfere with other anesthesia if necessary, and that it causes great comfort to the patient in labor. It seems contraindicated when labor pains are extraordinarily weak, when a patient is already depressed by hemorrhage or by infection, although in eclampsia its use seemed to be indicated. So far as could be observed no unfavorable results of any sort followed its employment.

Puerperal Lumbar Neuritis.—HAUCH (*Zeit. f. Geb. u. Gyn.*, Band lvii, H. 2, 1906) contributes a paper upon this subject, reviewing the literature and reporting cases of his own. In the Maternity Wards at Copenhagen in 680 parturient patients, neuritis was found in 32 (or 4.7 per cent.). The symptoms were pain on pressure over the nerves, paresis, pain in the nerves, hyperesthesia of the skin, and in some cases, increase of the patellar reflex. The nerves affected were mostly the crural and sometimes other nerves of the lower extremities. As typical of the condition, he describes the case of an unmarried primipara, somewhat anemic, who had suffered from prolonged nausea during pregnancy. She had also albuminuria. Labor was spontaneous; the child living, but the mother's recovery was complicated by great sensitiveness on pressure in the crural and saphenous nerves, with great pain on moving the lower extremities, hyperesthesia of the skin, and increased patellar reflex. The electric reaction was unchanged. The patient made a spontaneous recovery with no other treatment than rest in bed and good food. The author tabulates his thirty-two cases, describing the symptoms, course of labor, and giving other details. Sensibility to heat and cold was unchanged in all cases. Reaction to

electricity was also normal, and in but one case was there disturbance of the trophic or vasomotor function. There were varying paresis and disability in accordance with the generally depressed condition of the patient. It could not be observed that the size of the pelvis or the position or presentation of the child had any influence upon the occurrence of neuritis. In nine cases in the thirty-two the patient had nausea during pregnancy. This draws attention to the theory advanced that both nausea and neuritis are caused by toxemia. In five cases a mild influenza was present during the puerperal period. In thirty-one of the thirty-two cases labor was spontaneous. The condition seems one of neurasthenia, accompanying in many cases anemia and toxemia also. Hauch, however, reviews the pathology of the condition, and is inclined to believe that puerperal neuritis is the result of an acute intoxication which attacks the nerve filaments, but which lasts only a short time. He believes that various circumstances may make the inflammation better or worse, and that this accounts for the variation in the different cases. The diagnosis between neuritis and phlebitis is usually not difficult. In phlebitis the tenderness is usually in Scarpa's triangle; in phlebitis the veins are swollen and can be distinguished and there are œdema and fever; the patient frequently cannot move the limb and the pain does not extend along the nerve trunks, nor is the skin as sensitive as in neuritis. The diagnosis between hysterical paresis and neuritis is often difficult to make. The prognosis is good. Most patients recover in eight or ten days; occasionally one persists for two or three weeks. The treatment is entirely symptomatic and hygienic.

Extirpation of the Spleen during Pregnancy.—SCHAUTA (*Zentralb. f. Gyn.*, 1906, No. 25) reports the case of a pregnant patient, two months advanced, who had in addition a tumor close to the uterus. As pregnancy advanced the tumor could be separated from the womb and abdominal section was performed about the fourth month. It was found to be an enlarged spleen and was removed without especial difficulty by a transverse suprapubic incision. The patient made an excellent recovery. On the day of operation examination of the blood showed 5,350,000 red and 28,000 white blood cells. Eight days after operation the red cells were not especially altered and but slightly decreased in quantity, while the white cells had fallen to 16,600. Three months after operation the red cells were 3,860,000; the white, 14,900. Removal of the spleen is a rare operation during pregnancy and the statistics of the subject are very meager.

Frozen Section through the Body of a Parturient Patient Dying during the Expulsive Period of Labor.—BUMM and BLUMREICH (*Zeit. f. Geb. u. Gyn.*, 1906, Band lvii, H. 2) describe a recent frozen section made through the body of a patient dying in labor. The woman was a primipara, aged thirty-nine years, previously healthy, with practically normal pelvis, with the fœtus in normal position and presentation. After the membranes had ruptured and while the labor pains were continuing and the patient seemed in perfectly normal condition, she suddenly became cyanotic, with well-marked dyspnœa, and suddenly died. On examination the head of the child was deep in the pelvis, the occiput on the left side and anterior. The child's heart sounds

had ceased. Eleven hours after death the body was placed in a cold mixture and thoroughly frozen. On the sixth day sections were made in such a position as to open the genital tract anteroposteriorly. Photographs were then taken and the specimens were then placed in 4 per cent. formalin for two weeks. Examination of the specimen showed that below the contraction ring there was a considerable area of tissue covered by decidua, and then a portion 4 cm. long of cervical mucous membrane. The specimen showed very plainly a contraction ring whose lower portion was considerably dilated. The cervix, and the cervix only, extended from the vagina to the uterus proper, while above this the uterine muscle could distinctly be demonstrated, and undoubtedly was capable of contraction during labor.

Thrombosis of the Placental Vein, with Intrauterine Death of the Fœtus.—MATHES (*Zentralb. f. Gyn.*, No. 25, 1906) reports the case of a primipara, aged thirty-nine years. There was protrusion of the uterus and abdominal contents, and rachitic pelvis. The patient was admitted to the hospital in labor; but soon after her entrance fœtal heart sounds could not be heard. Labor lasted but seven and one-half hours, terminating spontaneously; the amniotic liquid was discolored when the membranes ruptured. The child had died but a short time before birth, as signs of maceration or change were absent. On examining the placenta the attachment to the umbilical cord was eccentric; the vessels of the chorion on the fœtal side were filled with blood, and one of the veins running to the cord was thick and greatly distended with blood. The placental tissue about the vein was infiltrated with blood and greatly thickened. The vein itself was hard, and when cut across, exuded dark blood and also threads of fibrin; the vein was then incised lengthwise, when it was found to be the site of thrombosis. Microscopic examination showed the characteristic structure of a thrombus and also the fact that the endothelium had disappeared from the inner aspect of the vein. On the inner layer of the muscular tissue there were also masses of leukocytes.

OPHTHALMOLOGY.

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The Refraction of the Newborn.—ELSCHNIG (*Zeitschr. f. Augenh.*, 1906, vol. x) has frequently found in children, two hours to two days after birth, a myopia from 4 to 7 D., disappearing after atropine, and

giving place to low hypermetropia. Such spasm of the accommodation may be due to an abnormal reaction of the eye to the first luminous excitations. Some newborn infants are hypermetropic or emmetropic at birth; in these cases the pupils are dilated and the eyeballs almost immobile.

Eumidrine (Nitrate of Methylatropine), a New Mydriatic.—DEL MONTE (reviewed in *Arch. d'Ophtal.*, July, 1906) brings forward a new mydriatic, nitrate of methylatropine. Upon healthy and diseased irides, it acts, in equal doses with atropine more slowly, with less energy, and its effect is not so lasting; on the other hand, it is less toxic and appears to have no influence upon the tension. It can be substituted for atropine in children, the aged, and when the latter is not well borne. In 0.5 per cent. solution it acts more rapidly than homatropine, but its effects last about ten hours longer; this can be decidedly shortened by pilocarpine, rendering eumidrine preferable when dilatation of the pupil is desired for ophthalmoscopic examination.

The Symptoms of Disease of the Cerebral Peduncles, Especially those Affecting the Eyes.—MOERCHEN (*Ztschr. f. Augenh.*, 1906, vol. x) emphasizes a characteristic symptom: Superior alternating hemiplegia or syndrome of Weber; namely, hemiplegia of the side opposite the lesion with paralysis of the oculomotor nerve of the diseased side. This syndrome was found in forty-one of fifty-three cases; the facial was also involved in ten cases, and the hypoglossal in three. The paralyses vary in degree, but ptosis upon one side with paresis of a member of the opposite side are sufficient to establish the diagnosis. Instead of paresis, trembling or choreic movements have been noted, Benedict's syndrome. Disturbances of speech are likewise pathognomonic of a lesion of the left cerebral peduncle, if they are accompanied by a superior alternating hemiplegia. The branches of the oculomotor are involved in frequency as follows: the elevator, internal rectus, superior rectus, inferior oblique. In most cases ophthalmoplegia interna is also present. The pupil is immovable to reflex stimulation in some cases. It is rare for the paralysis to affect the third pair in their entirety, but sometimes, when the lesion involves the nuclei, certain branches of both sides are affected. "Choked" disk is very rare; it has been found in only 6 per cent. The visual disturbances are due either to papillitis or to secondary compression of the optic tracts. All other symptoms which have appeared in published cases are only "phenomena of contiguity."

The Ocular Symptoms in Disease of the Temporal Lobes.—KRÜGER (*Ztschr. f. Augenh.*, 1906, vol. x) finds local symptoms pointing to disease of the temporal lobes as follows: (1) Disturbances of speech in 27 per cent. These depend frequently not upon a cortical lesion, but are due to a lesion of the associated tracts. (2) Disturbances of audition, explicable by the termination of the auditory nerve in the temporal lobes; deafness of both ears is found when an extensive sarcoma compresses both temporal lobes; tinnitus is also present. Olfactory disturbances are rare. The ocular symptoms observed are never symptoms of localization. Hemianopsia is present in 14 per cent.; it is due to a lesion of the optic fibers which traverse the lower region of

the temporal lobe. Conjugate reaction is rarely seen. Immobility of the pupils, optic atrophy, and neuroretinitis are found in 14 per cent. They show nothing characteristic. The disturbances of the pupil depend upon lesions of the optic nerve. Nystagmus has been observed occasionally. The paralyses of the motor nerves are due to secondary compression at the base.

DISEASES OF THE LARYNX AND CONTIGUOUS STRUCTURES.

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Unusual Motility of the Tongue.—TAPIA (*Revue hebd. de laryngologie, d'otologie, et de rhinologie,* July 7, 1906) reports a case in which the subject, a young student of medicine, could place his tongue into either posterior nasal passage, and could detect the orifice of the pharyngeal bursa and its prolongations into one median and two lateral recesses on either side—in all five depressions—in his hypertrophied pharyngeal tonsil. He could also feel with his tongue the anatomical details of his rhinopharynx, such as the posterior border of the septum, the ends of the turbinals, and the orifices of the Eustachian tubes, etc.

Velopalatine Angioma.—BAR (*Annales des maladies de l'oreille, du larynx, du nez, et du pharynx,* July, 1905) reports an example of this rare lesion in a rheumatic man, some fifty years of age. The growth was the size of a half-cherry and extended from the uvula, which it had enveloped, as far as the superior border of the right anterior pillar. It was treated by galvanopuncture, which it was hoped would be successful.

Primary Syphilitic Infection in the Nose.—JAMES T. CAMPBELL (*Annals of Otology, Rhinology, and Laryngology,* June, 1906) reports a case of primary syphilitic infection in the nose of a surgeon in perfect health who had circumcised a patient upon whose prepuce there was a large indurated chancre. The lesion occupied the anterior end of the right inferior turbinated body. The character of the infection was not recognized until the appearance of a macular rash upon the abdomen some eighty days after the probable inoculation. After six inunctions of mercurial ointment, one dram each at bedtime, all evidences of the disease had disappeared. Preceding the report of this case is a succinct summary of the bibliography on the subject; and likewise of its diagnosis.

Adenocarcinoma of the Nose.—FIOCRE (*Archives internationales de laryngologie, d'otologie, et de rhinologie,* Mai-Juin, 1906, p. 1094) reports an instance of adenocarcinoma of the nose, latent in evolution,

and without epistaxis or other premonitory symptoms, in a woman aged seventy-three years. It was attached only to the inferior and middle turbinals and was readily removed with gouge and forceps. Histologically, it was a myxo-adenoma with manifest tendency to carcinomatous evolution. No postoperative treatment was required. There was no recurrence.

The Importance of a Microscopic Examination of All Growths Removed from the Nares, together with a Report of Early Diagnosis of Malignant Growths.—SYLVAN ROSENHEIM (*Johns Hopkins Hospital Bulletin*, June, 1906), after intimating that intranasal tuberculosis and malignant neoplasms are more frequent than are usually known, and that, therefore, all growths removed from the nose should be systematically examined, reports six cases, two of which are illustrated, in evidence of the correctness of the importance of his views.

Adenosarcoma of the Nose.—FIOCRE (*Revue hebd. de laryngologie, d'otologie, et de rhinologie*, July 7, 1906) reports a case of adenosarcoma of the nose. The patient was a woman, aged sixty-three years, in whose right nasal fossa a tumor had developed at the expense of the middle and inferior turbinals. It was completely removed with forceps and gouge in three sittings. There had been no recurrence at the end of six months.

Osteocalcareous Growth in the Nose.—H. B. LEMERE (*Jour. Amer. Med. Assoc.*, July 28, 1906) reports a large osteocalcareous tumor in the nose of a girl aged fifteen years. It was removed with trephine and chisel after several days' work.

Epitheliosarcoma of the Ethmoid.—VACHER (*Annales des maladies de l'oreille, du larynx, du nez, et du pharynx*, Mars, 1906) reports a successful operation upon a man, aged seventy-nine years, for an epitheliosarcoma of the ethmoid, which had invaded the two frontal sinuses, the left sphenoidal sinus, and the left maxillary sinus.

Heredity in the Diseases of the Accessory Sinuses.—BICHATON (*Revue hebd. de laryngologie, d'otologie, et de rhinologie*, June 23, 1906) contributes a paper in which he contends that heredity is a great factor in the diseases of the accessory sinuses of the nose, and reports five cases in support of his contention.

Infectious Frontal Sinusitis with Local Necrosis and Pulmonary Gangrene.—CASTEX (*Revue hebd. de laryngologie, d'otologie, et de rhinologie*, June 23, 1906) reports a case of a young man recovering from scarlatina, complicated with nephritis, who sniffed up borated water to cleanse his nasal passages. Double frontal sinusitis supervened, requiring two operations, opening largely the two frontal sinuses and the ethmoidal labyrinth; two sequestra were removed at the second operation and the opening was further enlarged. Pulmonary gangrene with fœtid sputa set in eight days later. Colloidal silver was employed by friction and by injection, and recovery ensued.

DERMATOLOGY.

UNDER THE CHARGE OF

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The Relationship Between Hydroa Estivale and Hematoporphyrinuria.—LINSER (*Archiv. f. Dermatologie und Syphilis*, Band lxxix, Hefte 2 und 3) reports the following exceedingly interesting case observed in Romberg's clinic at Tübingen: A man, aged forty-four years, had suffered every summer since his sixth year from an eruption of vesicles situated upon the hands and face which disappeared in the winter, leaving numerous scars. These repeated attacks had produced a cicatricial condition of the entire face, with marked deformity of the ears, upper lip, nose, and fingers, the terminal phalanges of both index fingers being lost. (The patient had observed that the eruption was always most severe after exposure to the sun, and that his urine at this time was always dark, whereas, in the winter it was clear.) Upon his reception in the clinic there were present numerous small ulcers in the midst of the scar-tissue on the face and hands. After exposing the hands to the Röntgen rays for the purpose of making skiagrams, the urine became dark red and gave the characteristic spectrum of hematoporphyrin. Later the patient was exposed to the light of an electric arc lamp for some time, and this was followed by marked reddening of the face, with a burning sensation and the appearance of hematoporphyrin in the urine again. Exposure to hot air and to ordinary light had no effect upon the skin or urine.

Lymphadenomatous Prurigo.—DUBRENILH (*Brit. Jour. of Dermatology*, May, 1906, p. 197) refers to cases in the literature in which there occurred small and large lymphatic nodules accompanied with itching, without apparent leukocytosis in the majority of cases recorded. In some there was papular eruption upon the skin, of the form known as prurigo, in others there was merely intense itching without eruption. The author records two cases in which there was general enlargement of glands with intense itching and progressive wasting and loss of power. In one case the blood showed 1 white to 145 red corpuscles; of the white cells 90 per cent. were polynuclears. In the other cases there was also pronounced leukocytosis, with 91 per cent. polynuclears. The skin in the first case showed a number of lymphoid nodules, formed of cells resembling lymphocytes, with a nucleus, round or oval, separated by a very fine network of connective tissue. The sweat glands were much altered, the coils dilated, the lumen filled up with cells. The bloodvessels showed no changes. The skin was otherwise normal.

The Inclusions Found in Lupus Tissue.—PICK (*Archiv. f. Dermatologie und Syphilis*, Band lxxviii, Hefte 2 und 3) from a study of the

peculiar cell inclusions occasionally found in the tissue of lupus vulgaris concludes that these have nothing to do whatsoever with parasites, and that they especially are not blastomycetes, as Pelagatti believes. They probably arise from the deposition of lime and iron in and around resistant tissue elements of different kinds. In the case observed by Pick it was the lanugo hairs which served as the foreign bodies.

Effect of the Finsen Light upon Normal and Tattooed Skin.—Meirowsky-Graudenz (*Monatshefte f. Praktische Dermatologie*, Band, xlii, No. 8), as the result of his study of numerous specimens of human skin which had been subjected to the action of the light of the Finsen-Reyn lamp, concludes as follows: The light incites the epithelial cells to increased activity, which becomes evident through nuclear-division figures and the new formation of pigment. Longer action produces enormous injury to the epidermis which goes through a series of degenerative changes, finally becomes necrotic, and is elevated in blebs. Simultaneously with this injury a dilatation of the superficial and deep vessels appears, which has as a consequence an abundant emigration of leukocytes and hemorrhages into the tissues. The light likewise brings about an increase of the connective-tissue cells and a swelling of the collagen. After the inflammatory phenomena have reached their acme reparation begins with a marked increase in the thickness of the prickle-cell layer, whose cells, in the beginning without pigment, later show an increase and new formation of pigment in all the layers of the epithelium. In the connective tissue the œdema disappears, and there finally remains hyperpigmentation in the prickle-cell layer and a marked increase of the collagen. In the tattooed skin the Finsen light, by producing necrosis, makes possible the exit of pigment granules lying deeply in the corium.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

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Studies in Absorption.—Buxton and Torry (*Jour. Med. Research*, 1906, xv, p. 5) present an extensive study of absorption from the peritoneal cavity. The absorption of inert particles (lamp-black and red blood cells of chickens) and bacteria (typhoid bacilli) has been made the subject of a large number of experiments, the results of which are presented in five separate papers.

First, the subject of absorption of inert particles was studied by intraperitoneal injections of 3 c.c. of lamp-black suspensions. The

animals (guinea-pigs) were then killed and examined at various periods of time following the injections.

The peritoneal fluid was examined, and a leukopenia in the fluid occurred, which lasted from one to three or four hours. Then, in two to six hours, phagocytic leukocytes appear, and take up lamp-black rapidly; while in twenty-four hours, the mononuclear macrophages (large phagocytic cells, probably derived from connective tissue) appear. These latter cells play an important role in ingesting chickens' red cells; but it is a question whether their role is important with lamp-black.

On examining the spleens and livers of the guinea-pigs, it was found that the lamp-black reached these organs in fifteen minutes, when more was found than in animals killed thirty to sixty minutes after injection. In four hours, the amount present had greatly increased. This fluctuation in the amount of lamp-black present in the organs was seen to occur, so that at twenty-four hours, and again in four days, a maximum amount was present. An excretion and a renewal of particles must occur in the organs, but for this the authors offer no explanation.

Secondly, the absorption of typhoid bacilli from the peritoneum was examined. Rabbits were used. The animals were bled to death, in order to carry away the bacilli in the blood as far as possible; and the peritoneum was washed out with salt-solution at the time of death. The number of bacilli in the wash-water, various organs, and fluids was determined by plating. It was found that the number of bacilli in the wash-water and various organs varied greatly, and the time between the injections and examinations was not an index to their number. On the other hand, there may be an "explosive destruction" of a vast majority of the bacilli, few or none reaching the organs in such cases. The organs were practically sterile in three of seven rabbits examined ten to fifteen minutes after injection, the number in the wash-water being also greatly reduced, while bacilli swarmed in the organs and were in millions in the wash-water of the other four animals examined after the same length of time.

There may be a slower destruction, which is practically complete within an hour; but then large numbers of bacilli find their way into the circulation and are deposited by the blood in the various organs. The number of bacilli in the organs rapidly decreases after the first few minutes, up to about two hours after inoculation; but from seven to eight hours there is a considerable increase in their numbers, probably due to multiplication after the body fluids have expended all their destructive energy. From six to forty-eight hours there is again a decrease, probably mostly due to phagocytosis. Thus we see the variation in numbers of bacilli at varying lengths of time after injection follows the same changes as the inert particles. The bile and urine did not appear to be channels of excretion under the conditions of the experiments, *i. e.*, without lesions of liver and kidneys.

Thirdly, the function of the diaphragm in absorption is discussed. The authors found that an immediate rush of particles to the lymphatics of the diaphragm followed the intraperitoneal injections. The particles almost immediately reach the circulation by way of the lymphatics of the anterior mediastinum and thoracic duct, and so reach the organs in the earliest stages practically in a free condition. The particles are found, during the ten or eighteen minutes after injection, free in the lymphatic ducts and in the afferent ducts of the lymph glands; while later, they are all through the glands, largely within wandering cells. In the anterior,

mediastinal lymph nodes, the macrophages are the principal phagocytes; and the leukocytes play a comparatively minor part, not only with inert particles and animal cells, but also with bacteria. The authors are not able to decide whether the particles pass into the lymphatics through stomata, or whether they pass between the lining endothelial cells.

The fourth paper deals with the function of the omentum in absorbing inert particles. It is found that almost immediately after injection, fibrin accumulates on the omentum, and entangles particles and phagocytes. Here phagocytosis of lamp-black occurs in about ten minutes, and of chicken red cells in about one hour. If not overcrowded, the macrophages enter the omentum and appear as "trailers," or clasmatocytes, where the chicken red cells are soon ingested, while the lamp-black is retained for months. The macrophages are the most active agents of phagocytosis on the omentum, while the leukocytes play a minor part. The mesenteric lymph glands do not share in the initial rush of particles, such as is seen in the anterior mediastinal glands, and, to a less extent, in the omental glands.

The fifth paper consists of a discussion of the part played by the omentum in absorbing bacteria. The omentums of about ninety rabbits and seventy-five guinea-pigs were examined in this investigation. Typhoid bacilli became fixed in immense numbers to the omentum, like inert particles, by fibrin or over the "milky spots" seen in the omentum. Some bacilli may be in macrophages. The bacilli may be rapidly destroyed, both intracellularly and extracellularly; and do not reach the organs, as was seen in three of the seven cases examined immediately after injection. On the other hand, if there is a poor reaction of polynuclear leukocytes, the principal factors in rapid destruction, multiplication may occur "in vivo;" and so the increase in from four to six hours, before mentioned, is explained. These secondary centres of multiplication are not found in sixteen to twenty-four hours in animals that are recovering, while they are marked features if the animal is dying or dies between these times. As with inert particles, the macrophages are the chief factors in phagocytosis; but they cannot dispose of the bacteria by lysis without the presence of polynuclear leukocytes.

Experimental Neurotic Œdema of the Lungs.—Jores (*Deut. Arch. f. klin. Med.*, 1906, lxxxvii, 389) in attempting to find some explanation for certain types of œdema of the lungs, inserted one end of a small glass cannula into a branch of one of the main bronchi of a dog, and connected the other end of the cannula with a flask filled with carbon-dioxide. After twenty minutes, the dog was killed, and the lower lobe of the left lung, the area supplied by the cannula, was found to be markedly œdematous, while the remainder of the lung showed no marked alteration.

The experiment was repeated with various modifications, and it was found that the only explanation for the occurrence of œdema in this case seemed to be the mechanical irritation of the tube in the finer bronchi. Local asphyxia from aspiration of carbon dioxide or hindrance to respiration did not produce œdema; nor did interference with respiration, caused by section of the phrenic nerve or section of the cervical spinal cord, give rise to œdema. Closure of branches of the

bronchi, combined with compression of the pulmonary veins, likewise yielded negative results. Finally, irritation of localized areas of the lungs was resorted to, by means of a faradic current. The experiments were conducted both with and without a Sauerbruch's chamber. Stimulation by the faradic current of the surface of the lung produced definite œdema localized to the area irritated. Jores believes that the œdema is due to the stimulation of the vasomotor nerves by the electric current, and not to a direct action upon the musculature of the vessel-walls. This point does not, however, seem to be clearly established. The right vagus of a rabbit was severed, and three weeks later localized œdema of the right lung was produced by faradic stimulation of the surface. Jores argues that the vasomotor nerve may reach the lung through the sympathetic system, but the correctness of this supposition could not be proved; for all the animals operated upon died before the experiments could be completed. Section of both vagi at three to nine day intervals, with stimulation of the peripheral stump of the nerve first cut, occasionally gave rise to slight œdema of the lungs. Jores considers that a neuropathic type of lung œdema in man may be of importance in explaining certain varieties of this condition.

HYGIENE AND PUBLIC HEALTH.

UNDER THE CHARGE OF

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Bactericidal Action of Silver Compounds.—At the request of the Therapeutic Committee of the British Medical Association, C. R. MARSHALL and E. F. M. NEAVE have investigated the germicidal power of the various silver preparations, fifteen in number, in common use. As test objects were used a mixed culture obtained by allowing raw beef to stand from four to six weeks in water in an open vessel, and a pure culture of Staphylococcus pyogenes aureus. The conclusions arrived at (*Brit. Med. Jour.*, August 18, 1906, p. 362) are as follows: The experiments show that as regards bactericidal action the various silver compounds investigated fall into three groups: (1) Those which are powerfully bactericidal; (2) one, nargol—much less powerfully bactericidal; (3) two, argyrol and collargol—which possess practically no bactericidal action whatever. The first group includes silver nitrate, silver fluoride, actol, itrol, argentamine, argentol, albargin, argonin, ichthargan, largin, novargan, and protargol. The bactericidal action of these, in solutions containing the same percentage of combined silver, is closely similar, and it is practically impossible to place them in any order of activity which would be true under all circumstances. As argyrol and collargol are not bactericidal, it is evident that the amount of silver which a compound may contain is no criterion of its bactericidal power. Moreover, in view of the results obtained with argyrol, it seems

impossible to attribute the good effects which many clinicians have obtained with it to its bactericidal action.

The percentage of metallic silver in each preparation was determined in advance. The results follow: Collargol, 86.6; silver fluoride, 81.7; silver nitrate, 63.6; itrol, 60.8; actol, 51.5; argentol, 31.2; ichthargan 27.1; argyrol, 20; albargin, 13.4; nargol, 9.6; largin, 9.4; novargan, 7.9; protargol, 7.4; argentamine, 6.4; argonin, 3.8.

Infantile Mortality and Factory Employment of Women.—At the National Conference on Infantile Mortality, held in London, in June, 1906, GEORGE REID (*Lancet*, August 18, 1906, p. 423) contrasted the infantile mortality of two districts, identical in health conditions, but with the important difference, that, in the one in which the chief industry is making pottery, women are largely employed; whereas in the other, where mining and iron-working are the principal industries, there is practically no employment for women. From figures obtained from the Registrar-General, he classified the artisan towns into three groups: (1) Those in which the proportion of employed married and widowed females between the ages of eighteen and fifty years reached or exceeded 12 per cent. of the total number of women of those ages; (2) those in which the proportion was from 6 to 12 per cent.; and (3) those in which it was below 6 per cent. In group 1, the infantile mortality was highest and in group 2 it was lowest, and this order was found to obtain constantly since 1881. Thus, in the decades 1881–1890, 1891–1900, and four-year period 1901–1904, the average yearly infantile mortality rates of group 1 were 195, 212, and 193; of group 2 were 165, 175 and 156; and of group 3 were 156, 168, and 149. The reasons for these differences are not far to seek. The factory working-woman stays at her work until confinement and returns as soon afterward as possible, leaving her child to the care of others and to be fed artificially; the home-staying woman has far lighter work up to confinement and can give her infant personal attention and its natural food.

Naphthol Preparations in Disinfection.—Although both alpha- and beta-naphthol possess very marked bactericidal power, their very slight solubility in water has made them unavailable for general use; and attempts to introduce beta-naphthol in the form of its soluble sodium compound, which is much less powerful than free naphthol, have come to naught, because through the influence of light and air the compound is converted into an insoluble inert substance. SCHNEIDER (*Ztsch. f. Hygiene u. Infectionsk.*, 1906, lii, 534) found that equal parts of beta-naphthol and dried sodium carbonate, thoroughly mixed, can be employed to advantage, the carbonate promoting the solution of the naphthol in water without forming with it a new compound. A 1 per cent. solution of this mixture proved able to kill, in ninety-six hours, dried anthrax spores which resisted the action of 5 per cent. lysol for ten days, and a slightly stronger solution (1.50 per cent.) destroyed them in seventy-two hours. Against typhoid organisms and staphylococci the mixture proved to be about twice as powerful as lysol.

Typhoid Fever from Inhaled Infected Spray.—In a village in Alsace, one hundred persons were seized with typhoid fever within a period of six months. The infection is attributed by GARCIN (*Strassburger*

med. Zeit., January, 1906, p. 36) to water from an old well, which was used solely for feeding a spraying apparatus in a spinning room. The water was not available for drinking purposes, and hence, infection could have gained access to the system only through inhalation.

Detection of Typhoid Bacilli in Milk.—An outbreak of typhoid fever in Kolozsvar has been traced by D. KONRADI (*Centralb. f. Bakt.*, 1905, I Abt, xl, 31) to milk, the specific organism having been detected in two of thirty-three samples of milk examined. It appeared that the son of the dairyman had a mild typhoid, which did not prevent him from doing the milking and thus infecting the milk directly.

Typhoid Carriers.—According to LENTZ (*Klinisches Jahrbuch*, 1905, xiv, 475) about 4 per cent. of persons with typhoid fever become chronic carriers of the bacillus. He discovered one individual who continued to eliminate the organisms in the feces forty-two years after having had the disease. DOERR (*Centralb. f. Bakt.*, etc., Orig., 1905, xxiv, 624) quotes cases cited by others, in which the organisms were isolated from the gall-bladder seventeen and twenty years, respectively, after recovery. KUTSCHER (*Berl. klin. Woch.*, 1905, xlii, 1620) believes that the mild cases are especially dangerous. KAYSER (*Arbeiten aus dem kaiserlichen Gesundheitsamte*, 1906, xxiv, No. 1) says that he has detected six chronic carriers who were the cause of the disease in others, and that of 205 cases of the disease occurring in Strassburg, in 1904 and 1905, twenty-eight were traceable to "healthy" carriers. KLINGER (*Ibid.*) discovered twenty-three carriers in a period of a little more than two years. All yielded the organisms in the stools and eight in the urine; and twelve of the number had no typhoid history.

The Budde Process of Preserving Milk.—R. TANNER HEWLETT (*Lancet*, 1906, 209) believes that as conditions now exist, the use of moderate amounts of preservatives is preferable to that of dirty milk, swarming with bacteria; moreover, without the use of preservatives in hot weather, much of the London milk supply would be received in condition unfit for delivery. Employing fresh milk, he tested the efficiency of the Budde process in samples to which cultures of various organisms were added, including B. typhosus, B. paratyphosus, B. diphtheriæ, B. tuberculosis, B. anthracis (sporing), B. coli, B. dysenteriæ, B. subtilis, B. mycoides, and Penicillium glaucum, and found that the non-sporing organisms were destroyed, and the spore-bearers greatly reduced in number. Hewlett states that milk properly treated by this method is unchanged in odor, appearance, taste or otherwise, and that it will keep without apparent alteration for eight to ten days in hot weather, although at the end of the process the whole of the hydrogen peroxide is decomposed and no trace of it can be detected. In ordinary milk, the reduction in the number of bacteria amounts to more than 99.9 per cent.

(The Budde process appears to yield widely differing results in the hands of different experimenters, due probably to differences in the commercial preparations of hydrogen peroxide used, and also to the degree of bacterial contamination of the milk with which the tests are made.—C. H.)

Poisoning from Foods.—An outbreak of food poisoning, involving about sixty persons, is reported by Dr. W. G. WILLOUGHBY (*Public Health*, August, 1906, p. 626). The patients were mostly adults and included every person who had eaten of a consignment of brawn, and no others. The symptoms varied in intensity according to the amount eaten, and came on a few hours after eating. They included fever, pain, vomiting, and diarrhœa. Examination of the brawn showed freedom from preservatives and the common poisons, and the presence of B. enteritidis sporogenes, which, from the surface, had probably grown all through the preparation. Lubenau (*Centralblatt f. Bakt.*, Orig., 1906, I Abt., xl, p. 433) reports an extensive outbreak of violent gastroenteritis due to meat dumplings. About three-fourths of the 400 inmates of a sanitarium were seized with great suddenness, there being no premonitory symptoms. Examination revealed in uneaten portions a bacillus belonging to the group of peptonizing milk bacteria. The dumplings were made of a large piece of meat which was cooked after being four days in the ice-chest and then kept two days before being finally used. The process of cooking failed to kill the spores, which later developed and elaborated toxins in large amount. A family outbreak, in which seven persons were seized with violent symptoms, after a meal of sausage, is reported by A. CAHN (*Strassburger medizin. Zeitung*, January, 1906, p. 27). The symptoms included persistent vomiting, diarrhœa with slimy stools, and fever. Part of the victims had rapid pulse and some had enlarged spleens and rose spots. On the third day all had recovered except a servant, who went through what appeared to be a genuine typhoid. In each case the stools yielded B. paratyphosus, type B, as did the uneaten sausage.

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